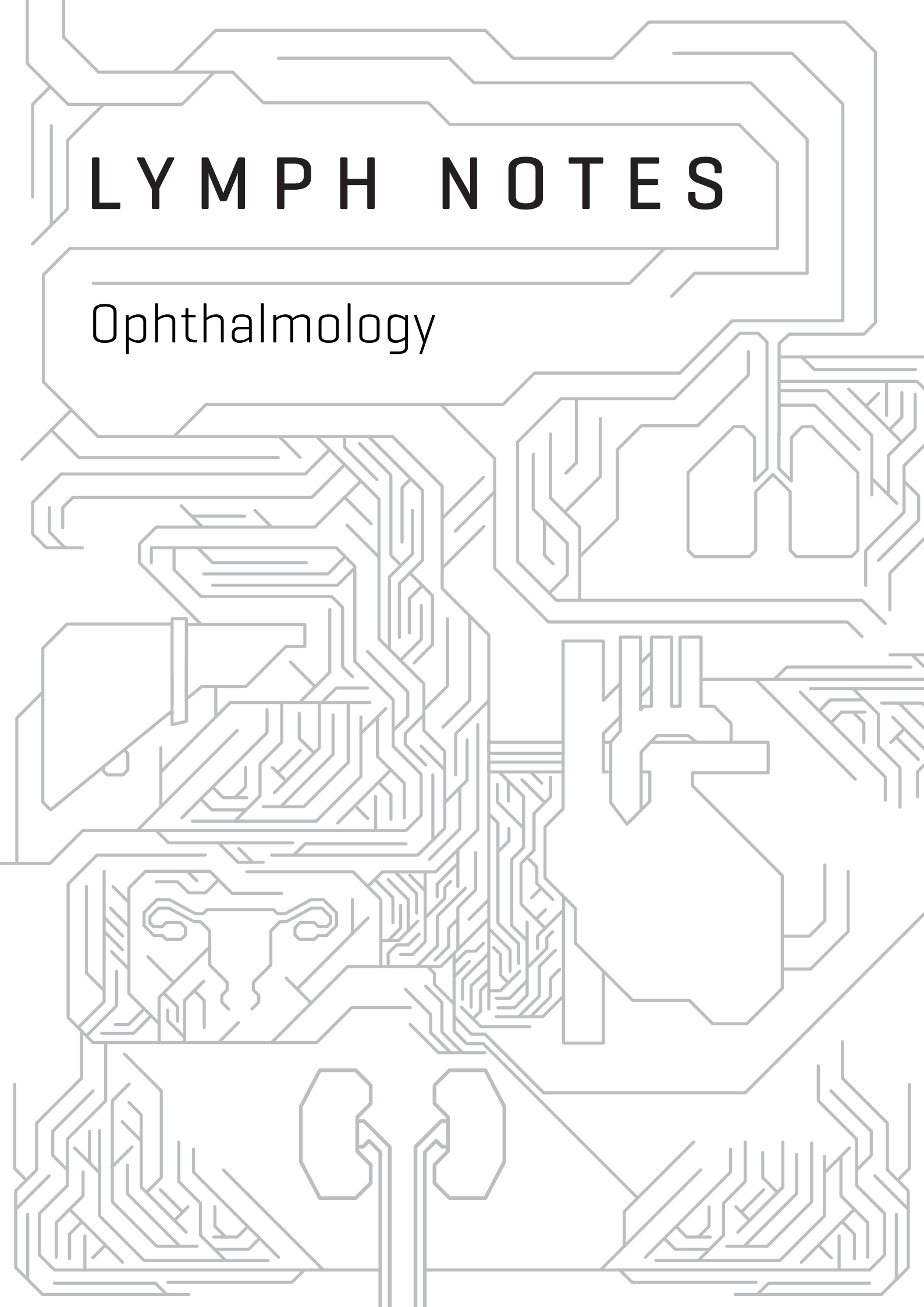


LYMPH NOTES

Ophthalmology



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Eye Lids

GIVE AN ACCOUNT

1. Describe a cross section of the upper lid. Illustrate your answer with diagram.

Draw with labels a section of the upper lid.

Describe the anatomy of the upper lid. Illustrate your answer with diagram.

Gross anatomy:

- the upper lid extends to the eyebrow while the lower lid passes without a demarcation line into the skin of the cheeks.
- In the primary position the upper lid covers 1/6 of the cornea(2mm) and the lower lid as the level of the lower limbus.
- The palpebral fissure: is an elliptical space enclosed between the two lid margins when the lids are open, in adults the fissure is 30 mm in length and 10-15 mm in width.
 - The canthi: are the angles of the palpebral fissure (rounded medially and acute laterally).
 - The upper lid crease: develops as the upper lid is raised due to the attachment of the levator muscle.

Minute anatomy (in sagittal section):

The eye lid is formed of 6 layers:

1. Skin:

Very thin skin, loosely attached to the underlying structures.

2. Subcutaneous areolar layer:

Loose connective tissue, containing no fat.

3. Muscular layer:

Containing the palpebral portion of orbicularis oculi muscle, the insertion of levator palpebrae superioris, & Muller's muscle.

4. Sub-muscular layer:

Loose connective tissue containing the main blood vessels & nerves of the lid.

5. Tarsus:

- a. The tarsal plate is a condensed fibrous tissue resembling cartilage.
- b. It acts as the skeleton of the lid.
- c. The end of the two tarsal plates are fixed to the orbital bones by the medial & lateral palpebral ligaments.
- d. 20-30 vertically-arranged Meibomian glands are embedded in the upper tarsus. They open in the lid margin, each by a single duct.

6. Palpebral conjunctiva:

- a. The conjunctiva is very thin, vascular, and firmly adherent to the tarsus by fibrous bands.
- b. Two millimeters above the lid margin, it shows a horizontal groove, the sulcus subtarsalis where foreign bodies are usually impacted

The lid margin (intermarginal strip):

It's the free margin of the lid being about 2mm broad and it's divided into lacrimal and ciliary parts.

The ciliary part has:

- **Anterior border** which is rounded and carries the eyelashes or cilia
- **Posterior border** which is sharp and in contact with the eye, its sharpness is necessary for conduction of tears towards the puncta.
- **White line:** corresponds to the openings of the meibomian glands.
- **Grey line:** which lines in the middle of the lid margin in front of the ducts of the meibomian glands. It corresponds to the submuscular space.

Glands of the eyelids:

1. Meibomian (tarsal) glands:

- Are sebaceous glands present in the substance of the tarsus (20-30 in the UL / 10-50 in the LL).
- They secrete an oily material which forms the outer layer of the tear film that prevents rapid evaporation of tears and lubricates the movement of the lid over the globe.

2. Zeis glands: Sebaceous glands related to the lash follicle.

3. Moll's glands: sweat glands related to the lash follicle.

Blood supply:

Arteries:

- **Medial palpebral arteries (superior and inferior)** from the ophthalmic artery.
- **Lateral palpebral arteries (superior and inferior)** from the lacrimal artery.
- **Branches from superficial temporal and facial arteries.**

Venous drainage:

Pre and post tarsal plexuses that drain into the ophthalmic veins.

Lymphatic drainage:

- Lateral 2/3 drains pre-auricular and parotid LN.
- Medial 1/3 drains to submaxillary LN.
- Then both in the deep cervical.

Nerve supply:

Motor: → The 3rd, 7th and sympathetic fibers supply the levator, orbicularis and muller's muscle respectively.

Sensory: → Ophthalmic division of trigeminal through supra-orbital, supraorbital (upper lid) and the infratrochlear nerves (lower lid).

Muscles of the lid include :

- **Striated muscles:** orbicularis oculi and levator palpebrae superioris.
- **Smooth muscles:** Muller's muscles.

Functions of the lids:

1. Protection of the eye from injury and excessive light by closure during sleep and blinking.
2. Prevent excessive dryness of the cornea and conjunctiva.
3. Tear secretion of the lipid layer.
4. Tear distribution by blinking.
5. Tear drainage by the sharp posterior border of lid margin.

2. Define blepharitis and enumerate its types

Definition:

Chronic inflammation of eye lid margin presents a chronic blepharitis

Types

- Squamous blepharitis
- Ulcerative blepharitis
- Parasitic blepharitis
- Angular blepharoconjunctivitis
- Allergic blepharoconjunctivitis

3. Give an account on ulcerative blepharitis:

Predisposing factor:

- Poor hygiene-Malnutrition
- Diabetes
- Errors of refraction

Exciting cause:

Staphylococcus aureus infection.

Clinical picture:

Symptoms

- Discharge
- Lacrimation
- Burning sensation
- Photophobia
- Itching
- Frequent falling of eye lashes

Signs

- Yellow crusts glue the lashes together
- Minute ulcers at the lid margin

- When the crusts are removed lid margin bleeds easily

Complication:

- Spread: chronic conjunctivitis, sty, marginal corneal ulcer
- Madarosis: due destruction of hair follicle
- Trichiasis: due to healing of ulcers by fibrosis
- Ptylosis: thickening and hypertrophy of the lid margin
- Epiphora: due to destruction of the sharp posterior lid margin which lead to eczema and thus etropion
- Ectropion
- Punctate keratitis: affecting the lower one third of the cornea
- Phlycten

Treatment:

General

- Improve general health
- Control DM if present
- Correct error of refraction

Local

- Lid hygiene
- Frequent massage to evacuate meibomian secretion
- Removal of scales by lid scrubbing by sodium bicarbonate or diluted baby shampoo.
- Elimination of the infection: Prolonged (2-3 weeks) rubbing of antibiotic ointment(gentamycin) into lid margin after removal of scales and crusts.

D.D:

- Mucopurulent conjunctivitis: dried discharge, on removal leaves an intact lid margin
- Squamous blepharitis

4. Give an account on chalazion

Definition:

Chronic nonspecific inflammatory granuloma of Meibomian gland

Etiology:

unknown and may be produced by

- Chronic inflammation by low virulence organism
- Retained contents of the gland (obstruction of the duct by proliferation of epithelium (Vit. A deficiency) or dry secretions
- The contents are irritant and excite granuloma around them
- The granuloma contains many giant cells

Clinical pictures:

Symptoms:

- Painless swelling along duration felt under the skin of the eye lid
- Pain occur only when of become infected (acute chalazion)

signs

- Slowly growing tenderness swelling of the tarsus.
- If the lid is everted the conjunctiva is seen red over the nodule with forced closure of the lids. Due to contraction of the orbicularis oculi muscle.
- It diminishes in size or disappears.

Fate and complication:

- Spontaneous resolution is rare
- Infection forming (acute chalazion)
- Marginal chalazion: the granulation tissue forms only in the duct and projects on the lid margin as a red nodule.
- Cyst formation
- It may open thorough conjunctiva
- It may reach a large size pressing on the globe and leading to astigmatism and mechanical ptosis
- Multiple chalazia may affect the whole tarsus

- Mechanical ectropion

Treatment:

- Very small chalazion:
Vit. A, local antibiotic and steroid preparation
- Marginal chalazion:
scraping from lid margin followed by diathermy
- Moderate or large chalazion:
vertical incision and scraping through conjunctival side (incision curette operation).
- Multiple chalazion:
combined excision of tarsus and conjunctiva leaving the lower third to avoid lid notching with replacement by mucous graft from the lid
- Recurrent chalazion of the same gland:
excision biopsy to exclude malignant tumour

5. Discuss Sty (External hordeolum):

Definition:

Acute Staph. Suppurative inflammation of sebaceous glands and sweat glands.

PDF (recurrence +/- multiplicity):

- DM
- Uncorrected error
- Staph. Belphearitis

Clinical picture:

painful swelling of the lids(dull aching then throbbing)

Diffuse stage:

Red-Hot-Tender-swollen lid.

Localized stage:

Pus pointing to skin along lash line in front of the grey line.

Complication:

1. Most serious: cavernous sinus thrombosis
2. Most common: Trachiasis

3. Recurrence & multiplicity

Treatment:

- **Diffuse stage:** Hot fomentations, systemic antibiotics and local antibiotic ointment and drops.
- **When pointing occurs:** the pus must be evacuated by : Epilation of the related lashes or **Horizontal** skin incision.
- **For recurrent cases:** correct the underlying causes.

6. Enumerate the congenital anomalies of the lid:

1. Epicanthus
2. Congenital ptosis
3. Blepharophimosis
4. Distichiasis
5. Coloboma of the lid
6. Ectropion
7. Entropion

7. Types of inflammations of the eye lid:

1. Preseptal cellulitis
2. Lid abscess
3. Lid glands: Acute Zeiss gland as sty or hordeolum externum/ Meibomian glands as hordeolum internum or chalazion.
4. Lid margin (anterior blepharitis): Ulcerative, Seborrheic,.....

COMPARE BETWEEN:

	<i>Hordeolum externum (stye)</i>	<i>Hordeolum internum (acute chalazion)</i>
<i>Site</i>	At the lid margin related to lash	In tarsus deep to orbicularis muscle
<i>Inflammatory signs</i>	Mild	Marked
<i>Swelling</i>	Related to lash	Yellowish spot of pus is seen shining through the palpebral conjunctiva
<i>Contraction of orbicularis muscle</i>	Not affected	Diminished
<i>TTT</i>	Antibiotics Horizontal excision to evacuate the pus	Antibiotics Vertical conjunctival incisors or horizontal skin incision and evacuation of pus

Eyelashes disorders

1. Enumerate disorders of the eyelashes:

1. Trichiasis
 2. Rubbing lashes
 3. Distichiasis
 4. Madarosis
 5. Poliosis
 6. Hypertrichosis
-

2. Describe the clinical picture, complications & treatment of senile (involutional) ectropion.

Clinical picture:

Symptoms:

1. Bad cosmetic appearance.
2. Epiphora, leading to eczema, leading to ectropion, leading to more epiphora & eczema.

Signs: depending on the degree:

1. Mild: exposure of the lower punctum.
2. Moderate: exposure of palpebral conjunctiva.
3. Severe: exposure of bulbar conjunctiva.

It's due to senile weakness of the orbicularis muscle and laxity of the palpebral ligaments.

Complications:

1. Loss of marginal strip of tears and epiphora.
2. Chronic conjunctivitis and xerosis.
3. Ulceration and opacification of the lower part of cornea.

Treatment:

A. Mild cases:

1. Instruct the patient to wipe his lower lid upwards.
2. Cautery to the conjunctiva: to induce fibrosis and contraction of the conjunctiva with correction of ectropion.
3. Snellen's inverting sutures to invert the lid.

B. Severe cases:

1. Lateral canthal sling; very effective to support the lower lid.
2. Horizontal lid shortening and blepharoplasty.

3. Comment on entropion.

Definition:

Rolling inwards of the eyelid margin towards the globe. The whole row of lashes will be rubbing against the cornea and finally there will be a deformity of the tarsus.

*Types:***1. Cicatricial (fibrotic) entropion:**

Fibrosis of the palpebral conjunctiva due to trachoma, chemical burns, diphtheria and ocular cicatricial pemphigoid.

2. Spastic entropion:

Due to spasm of orbicularis muscle in response to ocular irritation. e.g. inflammation, exposed sutures, operations following enucleation or enophthalmos. It may be temporary or permanent.

3. Involutional (senile) entropion:

Affects only the lower lid due to overriding of the preseptal portion of orbicularis muscle over the pretarsal portion.

4. Congenital entropion:

Usually affecting the whole lower eyelids. It should be differentiated from epiblepharon that affects the medial aspect with absence of lower lid crease.

*Clinical picture:****Symptoms:***

1. Foreign body sensation.
2. Photophobia.
3. Lacrimation.
4. Blepharospasm.

*Complications:***1. Conjunctival:**

- a. Chronic conjunctivitis.
- b. Conjunctival ulcer.
- c. Epithelial plaque.

2. Corneal:

- a. Recurrent ulceration, leading to corneal opacities.
- b. Superficial vascularization.
- c. Epithelial plaque.

*Treatment:***1. Cicatricial entropion:**a. In the upper lid:

- Snellen's operation: removal of a wedge of the tarsus. The edges are approximated by sutures, everting the lid.
- Webster's operation.
- Anterior lamellar reposition.
- Transverse tarsotomy with marginal rotation.

b. In the lower lid:

- Webster's operation.

2. Spastic entropion:

- a. Treatment of any cause of irritation. e.g. treatment of a corneal ulcer.
- b. Mild cases:
 - i. T-shaped plaster: This method is ineffective as it becomes loose.
 - ii. Lateral canthotomy: To weaken Riolan's muscle temporarily.
- c. Recurrent cases:
 - i. Lateral canthoplasty: to weaken Riolan's muscle permanently.
 - ii. Skin and muscle operation: where an elliptical area of skin and orbicularis muscle of the lower lid is removed. (This operation can be used in congenital entropion).

3. Involutional entropion:

- a. Everting sutures: temporary treatment in mild cases.
- b. Jones procedures: tucking of lower lid retractors.
- c. Lateral canthal sling: fixation of lateral part of tarsus to lateral orbital rim (very effective).

4. Discuss the types, clinical picture, complications and treatment of ectropion.*Definition:*

Rolling outwards of the lid margin from the globe. Usually affects the lower lid as it stands against gravity.

Types:

- 1. Involutional (senile) ectropion → due to weakness of the orbicularis muscle and relaxation of the palpebral ligaments.

2. Cicatricial (fibrotic) ectropion:→ due to scarring and contracture of the skin of the lower lids by burns, trauma or tumor.
3. Paralytic ectropion:→ due to paralysis of orbicularis muscle in facial nerve palsy.
4. Mechanical ectropion:→ due to increased weight of lower lid by e.g. multiple chalazion.
5. Congenital ectropion, rare.

Clinical picture:

Symptoms:

1. Bad cosmetic appearance.
2. Epiphora, leading to eczema, leading to ectropion, leading to more epiphora & eczema.

Signs: depending on the degree:

1. Mild: exposure of the lower punctum.
2. Moderate: exposure of palpebral conjunctiva.
3. Severe: exposure of bulbar conjunctiva.

It's due to senile weakness of the orbicularis muscle and laxity of the palpebral ligaments.

Complications:

1. Loss of marginal strip of tears and epiphora.
2. Chronic conjunctivitis and xerosis.
3. Ulceration and opacification of the lower part of cornea.

Treatment:

1. Senile ectropion:

a. Mild cases:

- i. Instruct the patient to wipe his lower lid upwards.
- ii. Caution to the conjunctiva: to induce fibrosis and contraction of the conjunctiva with correction of ectropion.
- iii. Snellen's inverting sutures to invert the lid.

b. Severe cases:

- i. Lateral canthal sling: very effective to support the lower lid.
- ii. Horizontal lid shortening and blepharoplasty.

2. Cicatricial (fibrotic) ectropion:

- a. Small scar: V to Y plasty or Z plasty.
- b. Large scar: skin graft (donor sites: post auricular area or upper inner arm).

3. Paralytic ectropion (facial palsy):

- a. Protection of the cornea: by drops during day and ointment during sleep.

- b. Medical ttt: to help nerve regeneration by cortisone anti-rheumatic medication & massage.
- c. Lateral tarsorrhaphy: to induce adhesion between upper & lower lids at the lateral canthus either temporary or permanent to narrow the palpebral fissure.
- d. Fascia lata sling and silicone sling operations: In sever & recurrent cases, where a sling is passed between the medial and lateral palpebral ligaments to support the lower lid in the normal position,

4. Mechanical ectropion: → Treat the cause.

5. Give a short account on Trichiasis:

Definition:

The condition where more than 4 lashes are rubbing against the cornea and conjunctiva

Etiology:

1. Congenital trichiasis:

Often in all 4 lids. Its's called districhiasis. In this condition, an extra row of lashes is present behind the gray line in the place of the ducts of the Meibomian glands.

2. Acquired trichiasis:

Maybe caused by the following:

- a. Trachoma is the commonest cause; due to fibrosis distorting the hair follicle.
- b. Ulcerative blepharitis.
- c. Burns.

Symptoms:

- a. Foreign body sensation.
- b. Photophobia.
- c. Lacrimation.
- d. Blepharospasm.

Complications:

1. Conjunctival:

- a. Chronic conjunctivitis.
- b. Conjunctival ulcer.
- c. Epithelial plaque.

2. Corneal:

- a. Recurrent ulceration leading to corneal opacities.
- b. Superficial vascularization.

c. Epithelial plaque.

Treatment:

1. Rubbing lashes: When 4 lashes or less are rubbing, ttt is to permanently destroy the hair follicle by:

- a. Thermal coagulation by diathermy.
- b. Chemical coagulation by electrolysis; due to release of NaOH.
- c. Cryocoagulation.
- d. Epilation: Pulling out of the lash is not a permanent treatment since the lash grows again in 4-6 weeks.

N.B:

Electrolysis:

- The +ve electrode is a wet pad of cotton applied to any part of the body.
- The -ve electrode is a needle introduced into the hair follicle.
- The current is switched 2-5 mA for 10 sec. Chemical coagulation of the hair follicle and the lash can be easily pulled out.

Diathermy:

- One electrode (a broad piece of lead) is tied to any part of the body.
- The second electrode is a needle, which is introduced into the hair follicle.
- The current is switched to 30 mA for 3 seconds.
- The follicle is coagulated and destroyed; the hair follicle and the lash can be pulled out easily.

2. If a group of lashes more than 4 are rubbing:

a. In the upper lid: Van Millengen's operation:

The principle is to displace the rubbing lashes away from the cornea by placing a buccal mucous graft in the gray line.

b. In the lower lid: Webster's operation:

The principle is to straighten the tarsus and lengthen the palpebral conjunctiva by placing a buccal mucous graft in an incision in the sulcus subtarsalis.

Transpositioning by Z-plasty can also be done

6. Discuss Ptosis:

Definition:

Drooping of the upper lid that normally covers the 2mm (or 1/6 of the cornea)

Etiology and clinical picture:

Congenital: due to levator dystrophy

- **Associations:**

1. Superior rectus palsy → hypotropia, limited.
2. Blepharophimosis:
 - Ptosis
 - Epicanthus
 - Lower lid ectropion
 - Narrow fissure
3. Marcus gun jaw winking phenomenon: due to 3rd & 5th

Nerves synkinesis → elevation of ptotic lid with jaw movement (pterygoid).

4. Absent lid crease
5. Lid lag on downgaze (levator unable to relax).
6. Errors of refraction.

- **Consequences:** if severe

1. Over action of the frontalis muscle → Elevation of the eye brows with an astonished look.
2. Compensatory head and body posture e.g. Chin elevation
3. Amblyopia

Acquired

1. Mechanical: e.g. chalazion
2. Myasthenia gravis: intermittent at night or with fatigue in middle aged female.
3. Aponeurotic:
 - Senile (involutional): usually bilateral
 - Surgical: damage of levator – superior rectus.

4. Third nerve palsy: severe, dilated pupil, divergent motility and poor levator function.
5. Horner syndrome: Mild, constricted pupil and good levator function

Complications:

1. Amblyopia in severe cases
2. Torticollis & Contracture of sternomastoid muscle

Treatment:

Congenital:

Factors affecting line of treatment and prognosis:

1. **degree of ptosis**
2. **bilaterality:** severe and bilateral ptosis should be treated early to prevent amblyopia.
3. **Timing:**
 - **Severe:** Immediate for the fear of amblyopia
 - **Not severe:** pre-school (<6 years) to avoid shyness or inferiority.
4. **Associated feature:**

Associated squint should be treated first to avoid postoperative diplopia.

Also, another congenital anomaly e.g: epicanthus should be treated first.
5. **Type of surgery: According to levator function & degree of ptosis:**
 - **Mild degree of ptosis with good levator function:** Levator tucking
 - **Moderate degree of ptosis with fair Levator function:** Levator resection.
 - **Severe degree of ptosis with poor levator function:** Frontalis or brow suspension (or tarsal sling).

Acquired:

1. **Mechanical ptosis:** treat the cause.
2. **Myasthenia gravis:** prostigmine or ptosis props.
3. **Paralytic ptosis due to 3rd nerve palsy:** wait for 6 months to allow regeneration then do surgical treatment. Associated squint should be treated first to avoid postoperative diplopia.
4. **Horner's syndrome:** fasanella servat operation (transconjunctival tarso-mullerectomy).

N.B:

- In MG jaw winking: bilateral sling operation.
- Contraindication of ptosis surgery: corneal hyposthesia and absent bell's phenomenon (10%) for the risk of exposure keratitis after surgery(lagophthalmous).

7. Discuss lagophthalmos:*Definition:*

Incomplete closure of the lids.

*Causes:***Local causes:**

1. Physiological: lack of orbicularis tone during sleep.
2. Paralysis of orbicularis e.g: VII or bell's palsy (paralytic ectropian).
3. Ectropian.
4. Exophthalmos (proptosis)
5. Scarring of the lid e.g. shortening after multiple snellen's operation
6. Symblepharon: adhesion between the bulbar and palpebral conjunctiva.
7. Coloboma of the lid.

General causes:

1. Severe illness and weakness with atony of the muscle
2. Coma.

Clinical picture:

In mild cases: →Lagophthalmos can be overcome by forcible lid closure.

In severe cases: →The eyes are permanently opened leading to:

- Epiphora
- Chronic conjunctivitis and xerosis
- Exposure or lagophthalmic ulcer: exposure of the cornea during sleep leads to dryness, ulceration and keratitis of the lower third.

Treatment:

1. Of the cause e.g: VII palsy.
2. Protection of the cornea during the day by contact lens or glasses & during sleep by applying ointment at night.
3. Lateral tarsorrhaphy: inducing adhesion between the lid margins.

N.B: the upper part of the cornea is protected by the UL as the eyes roll up during sleep. (bell's phenomenon).

8. Discuss symblepharon:*Definition:*

Adhesions between palpebral & bulbar Conjunctiva or Adhesions between eyelid and the globe (due to the presence of raw surface)

Aetiology:

Conjunctival scarring:

1. Chemical injuries and burns
2. Post-inflammatory e.g. trachoma, diphtheria,
3. Ocular cicatricial pemphigoid
4. Postoperative e.g. multiple pterygium surgeries

Types:

1. Anterior: between lids and cornea or bulbar conjunctiva
2. Posterior: between or at the fornix e.g. trachoma
3. Total: between bulbar and palpebral conjunctiva

Clinical picture: (3D)

1. Bad cosmetic appearance
2. Diminution of vision
3. Binocular Diplopia & limitation of mobility

Complications:

1. Exposure keratitis & chronic conjunctivitis.
2. +/- Ankyloblepharon

*Treatment:***A. Prophylactic ttt:**

1. Lubrication by steroids if no infection / if infected → antibiotic ointment

2. Contact sclera shell
3. Glass rod coated with antibiotic ointment between lid and globe

B. Definitive ttt:

1. Synechotomy
2. Mucous membrane graft
3. Keratoplasty (if cornea affected)

MCQS:

1. The lid sphincter muscle has the following portions:

- a. palpebral and orbital portions.
- b. Horner's muscle and riolan's muscle
- c. All of the above.
- d. None of the above.

Answer: (c) All of the above

2. Levator resection operation should be used in:

- a. Mild ptosis with levator function.
- b. Moderate ptosis with good levator function
- c. Moderate ptosis with poor levator function.
- d. severe ptosis with poor levator function.

Answer: (C) moderate ptosis with poor levator function.

3. Treatement of hordeolum internum include all of the following except:

- a. Hot fomentation
- b. Antibiotics
- c. Evacuation
- d. Epilation of the related lash.

Answer: (D) Epilation of the related lash

4. Inflammation of lid gland include all the following except:

- a. Ulcerative blepharitis
- b. Hordeolum extremum
- c. Hordeolum internum
- d. Squamous blepharitis

Answer: (A)Ulcerative blepharities

5. Entropion of lid margin is due to either spasm of orbicularis muscle or cicatricial contraction of palpebral conjunctiva:

- a. True.
- b. False.

Answer: (A) True

6. Trichiasis is caused by the following:

- a. Angular blepharitis.
- b. Hordeolum internum.
- c. Ulcerative blepharitis.
- d. Follicular trachoma.

Answer: (B) Hordeolum internum

7. Congenital ptosis is characterized by:

- a. Absent lid crease
- b. Chin elevation
- c. Forehead corrugation
- d. All of the above

Answer: (D) All of the above

8. Ptosis can occur in the following clinical situations:

- a. Following blunt trauma of the eyelid
- b. As a congenital condition
- c. In myasthenia gravis
- d. All of the above

ANSWER: (D) All of the above

9. Xanthelasma can occur in:

- a. Hypolipidemia
- b. Hyperlipidemia
- c. Hypoglycemia
- d. Hypocalcemia

ANSWER: (B)Hyperlipidemia

10.The commonest sign of Graves' disease :

- a. Exophthalmos.
- b. Lid retraction.
- c. Diplopia.
- d. Peri orbital edema.
- e. Conjunctival chemosis.

ANSWER: (B) Lid retraction

11.Ectropion of upper eyelid may be :

- a. Senile.
- b. Paralytic.

- c. Congenital.
- d. None of the above.

ANSWER: (D) None

12.All of the following are causes of lagophthalmos except :

- a. Facial N. palsy
- b. Proptosis.
- c. Cicatricial ectropion.
- d. 3rd nerve paralysis

ANSWER: (D) 3rd nerve

13.Ectropion of the upper lid most commonly :

- a. Spastic.
- b. Senile.
- c. Paralytic.
- d. Cicatricial

ANSWER: (D) Cicatricial

14.Tarrsoraphy is essential in :

- a. Bacterial corneal ulcer.
- b. Viral corneal ulcer.
- c. Exposure keratopathy.
- d. Traumatic corneal ulcer.

ANSWER: (C) Exposure keratopathy.

15.Superior tarsal muscle (Muller's muscle) is supplied by the :

- a. Third cranial nerve
- b. Sympathetic nerve fibres
- c. Parasympathetic nerve fibres
- d. Seventh cranial nerve

ANSWER: (B)

16.The anterior most structure in the eyelid margin is the :

- a. mucocutaneous junction
- b. gray line
- c. meibomian gland orifices
- d. lash line

ANSWER: (D)

17.The anterior lamella of eyelid contains :

- a. Glands of Wolfring
- b. Zeis glands
- c. Glands of Krause
- d. Meibomian glands

ANSWER: (B)

18.Which of these is a common occurrence with chalazia :

- a. Complete spontaneous resolution with time
- b. Conjunctival side of the lesion is reddish or purplish
- c. Transformation to malignancy
- d. Presentation as nodule in the intermarginal strip

ANSWER: (B)

19.The anterior and posterior lamellae of the lid can be separated at the level of the lid margin by the :

- a. lash line
- b. line of meibomian gland orifices
- c. gray line
- d. mucocutaneous junction

ANSWER: (C)

20.Tylosis refers to :

- a. loss of lashes
- b. misdirection of lashes
- c. blocked meibomian orifices
- d. hypertrophied drooping lid

ANSWER: (D)

21.Digital pressure on the lid margin leads to a pasty discharge from the meibomian gland orifices. Which of the following statements is TRUE regarding this patient :

- a. Epiphora is a common symptom
- b. Treatment with tetracyclines for 2 weeks is curative
- c. Punctate fluorescein staining of the ocular surface could be present
- d. Tear film break up time (TBUT) will be prolonged

ANSWER: (C)

22.Madarosis may be seen in :

- a. eyelid neoplasm
- b. chronic blepharitis
- c. Hansen's disease
- d. all of the above

ANSWER: (D)

23.Fibrin collarette around the base of the eyelashes in children is due to :

- a. squamous blepharitis
- b. meibomian seborrhea
- c. ulcerative blepharitis
- d. meibomianitis

ANSWER: (C)

24.Chalazion :

- a. is also called as tarsal cyst
- b. can result in preseptal cellulitis if untreated
- c. heals if the affected lash is pulled out
- d. is a non-suppurative inflammation of a Zeis gland

ANSWER: (A)

25.Lid findings in Stevens Johnson syndrome includes :

- a. distichiasis
- b. trichiasis
- c. irregular posterior margin
- d. All of the above

ANSWER: (D)

26.The vertical tautness of the lid can be restored in involutional entropion by :

- a. shortening the width of the tarsal plate
- b. lateral tarsal strip procedure
- c. reattachment of retractors to tarsal plate
- d. all of the above

ANSWER: (C)

27.Which of the statements below regarding both involutional type of entropion & ectropion is FALSE :

- a. Lid laxity is an important causative factor
- b. Lateral tarsal strip procedure attaching tarsus to lateral orbital rim is useful in both cases
- c. Tearing is a chief symptom
- d. Conjunctival spindle excision below the punctum can help mild cases

ANSWER: (D)

28.Cicatricial ectropion can result from all of the following EXCEPT :

- a. chalazion treatment
- b. burns
- c. trauma
- d. eyelid skin incision

ANSWER: (A)

29.Gold weight is placed pretarsally in the upper lid in :

- a. ankyloblepharon
- b. involutional ectropion

- c. lagophthalmos VII nerve palsy (Bell's palsy)e
- d. spastic entropion of upper lid

ANSWER: (C)

30.Causes of spastic enropion include

- a. Ocular bandaging
- b. Chronic blepharitis
- c. Stevens-Johnson syndrome
- d. All of the above

ANSWER: (A)

31.Abnormal lid laxity is diagnosed if :

- a. lid can be drawn away by more than 10 mm from the globe
- b. the punctum is visible only when the lid is pulled down
- c. pulling the lower lid laterally causes medial canthus displacement more than 4 mm
- d. the lid does not snap back immediately when drawn away from the globe and released

ANSWER: (D)

32.Keratinization of the lid margin can result from :

- a. Blepharospasm
- b. Severe ectropion
- c. Spastic entropion
- d. Lagophthalmos

ANSWER: (B)

33.The lower lid is kept opposed to the globe by which of the following mechanisms :

- a. The capsulopalpebral fascia acts as the lower lid retractor
- b. The tarsal plate is attached to the bony orbit via taut canthal ligaments
- c. The pretarsal orbicularis has firm connections with the tarsus
- d. All of the above

ANSWER: (D)

34. A male patient was complaining of continuous redness of both eyes, foreign body sensation, and frequent loss of lashes. On examination, the lid margins were hyperaemic, and the lashes were matted with yellow crusts, which left painful ulcers on trying to move. The most reliable diagnosis is

- a. Scaly blepharitis
- b. Cicatricial entropion
- c. Spastic entropion
- d. Ulcerative blepharitis

ANSWER: (D)

35. Staphylococcal infection of sebaceous glands of the eye lids is called

- a. Blepharitis
- b. Conjunctivitis
- c. Keratitis
- d. Hordeolum

ANSWER: (D)

36. Which of the following statements is most correct:

- a. Hordeolum involves the meibomian glands whereas Styes and Chalazions do not.
- b. Chalazions are caused by infections (usually Staphylococcus aureus) whereas hordeolums are not.

- c. Chalazions are often painless
- d. Hordeolums are bilateral and styes are unilateral
- e. All of the above are correct

ANSWER: (C)

37.True or False? The two major types of ptosis are congenital and acquired.

- a. True
- b. False

ANSWER: (A)

38.The most common type of congenital ptosis is :

- a. Aponeurotic
- b. Neurogenic
- c. Traumatic
- d. Myogenic

ANSWER: (D)

39.Extraocular movement testing in congenital myogenic ptosis may reveal limited :

- a. adduction
- b. abduction
- c. supraduction
- d. infraduction

ANSWER: (C)

40.True or False? Acquired ptosis is divided into

myogenic,neurogenic,aponeurotic,mechanical,protective and pseudoptosis causes.

- a. True
- b. False

ANSWER: (B)

41.Regarding Horner's syndrome

- a. Ptosis of moderate to severe degree may be seen
- b. The miotic pupil constricts to light
- c. Loss of accomodation is seen in the case of third order neuronal lesions
- d. The near reflex is absent in the miotic pupil

ANSWER: (B)

42.Pupil involving third nerve palsy is caused in case of

- a. intracranial aneurysm
- b. diabetes mellitus
- c. hypertensin
- d. all of the above

ANSWER: (A)

43.The position of the eye ball in a patient with total III nerve palsy is

- a. down & in
- b. up & in
- c. down & out
- d. up & out

ANSWER: (C)

44. A 4 year old child with severe bilateral congenital myogenic ptosis with poor elevator function

- a. will have a chin up head position
- b. will benefit from bilateral frontalis suspension procedure
- c. is likely to have amblyopia
- d. All of the above are true

ANSWER: (A)

45. Which of these acquired ptosis can be an indication of life-threatening condition

- a. ptosis with deep sulcus and lid drop in down gaze
- b. ptosis with pupil sparing III nerve palsy in a 50 year old diabetic
- c. ptosis due to isolated third nerve palsy following old closed head injury
- d. ptosis which increases by evening

ANSWER: (D)

46. True or False? Enophthalmos and microphthalmia are included in congenital ptosis.

- a. True
- b. False

ANSWER: (B)

47. True or False? Lid tumors and cicatricial conjunctivitis are included in mechanical acquired ptosis.

- a. True
- b. False

ANSWER: (A)

48.Components in blepharophimosis include

- a. horizontally elongated palpebral aperture
- b. bilateral ptosis
- c. epicanthus
- d. all of the above

ANSWER: (A)

49.All of the following statements regarding dermatochalasis are true EXCEPT

- a. It commences during puberty
- b. It results in pseudoptosis
- c. Blepharoplasty can correct the disorder
- d. There is redundant skin around the eye lids

ANSWER: (A)

50.All of the following types of entropion are known except

- a. Spastic entropion
- b. Senile entropion
- c. Paralytic entropion
- d. Cicatricial entropion

ANSWER: (C)

51.Third cranial nerve innervates all of the following except

- a. Superior oblique muscle
- b. Levator palpebrae muscle
- c. Inferior oblique muscle
- d. Medial rectus muscle

ANSWER: (A)

Conjunctiva

DISCUSS:

1. The gross and microscopic anatomy of conjunctiva

Gross anatomy:

Conjunctiva can be divided into the following parts:

1. Palpebral Conjunctive:

It lines the posterior surface of the lid and is formed of:

a) Marginal part:

- Starts at the grey line of the lid margin
- Continues to the sulcus subtarsalis.

b) Tarsal part:

- Very vascular
- Adherent to the tarsus.

2. The conjunctival fornix:

- It is the reflected part between the lids and the globe.
- It's formed of superior, inferior, medial and lateral fornices.

3. Bulbar conjunctiva:

- Covers the anterior part of the sclera
- Ends at the limbus

4. PlicaSemilunaris:

- Delicate vertical crescent-shaped fold at the medial canthus corresponding to a

rudimentary third lid seen in many animals

5. Caruncle:

- Small wart-like red structure
- present at the inner canthus
- with certain skin characteristics including fine hairs and sebaceous glands

Minute anatomy:

Conjunctiva is formed of 2 layers:

1. Anterior stratified columnar epithelium:

- It's 2-7 layers thick depending on the part of the conjunctiva
- contains numerous unicellular mucous glands (goblet cells) that secrete the inner mucoid layer of the tear film

2. The substantiapropria:

It's formed of:

a) The superficial adenoid layer:

- formed of fine CT rich in lymphocytes with no true lymphoid follicles
- It develops 3 months after birth

b) The deep fibrous layer:

- Thick meshwork of CT
- housing the nerves and vessels of conjunctiva

2. Causes, clinical picture and treatment of dry eye

Etiology:

1. Idiopathic atrophy of the main and accessory lacrimal glands
2. Congenital absence of the lacrimal glands
3. Keratoconjunctivitis sicca:
 - autoimmune disease leading to atrophy and fibrosis of the lacrimal gland
 - It occurs usually in females
 - and maybe associated with arthritis and dry mouth (Sjogren's syndrome)

- Other autoimmune destruction of the lacrimal gland, occurs with collagen vascular diseases
4. Medications that suppress tear secretion:
 - such as anticholinergics and antihistaminics
 5. Xerosis:
 - A condition where conjunctiva becomes thickened and loses its luster and transparency
 - the epithelium may become keratinized causing white foamy patches known as Bitot spots
 - In severe cases, scarring produces symblepharon
 - Tear deficiency occurs due to:
 - a) loss of goblet cells and accessory lacrimal glands
 - b) obstruction of the ducts of the main lacrimal gland
 - Causes of xerosis include:
 - a) vit. A deficiency
 - b) post inflammatory scar as a sequel of trachoma, chemical and thermal burns
 - c) Stevens-Johnson syndrome
 - d) ocular pemphigoid
 - e) irradiation
 - f) lagophthalmos
 6. Inflammation of lacrimal gland as sarcoidosis
 7. Tumors of lacrimal gland as mixed lacrimal gland tumor
 8. Conjunctival scarring due to:
 - a) Trachoma
 - b) chemical burns
 - c) Stevens-Johnson syndrome
 - d) Ocular cicatricial pemphigoid

Clinical Picture

ure

a) Symptoms:

- Irritation
- Discomfort
- Itching
- Burning
- Foreign body sensation

b) Signs:

1. Deficient precorneal tear film
2. Punctate epithelial erosion of the cornea
3. Watery (paradoxical reflex tearing) or mucoid discharge
4. Loss of corneal luster. In severe cases, corneal opacification with loss of vision occurs

Investigations

1. Tear film break – up time (BUT) with fluorescein is diminished (normally it's 15 seconds).
2. Schirmer test:
 - A normal person wets 10-30 mm. of a Whatman number 41 filter paper strip (5 mm. wide x 30 mm. long) in 5 minutes. Values less than 5 mm. indicate hyposecretion.
3. Rose Bengal staining of devitalized epithelial cells

Treatment

1. Treat the cause
2. Protective glasses and contact lenses
3. Replacement with artificial tears and lubricants
4. Preservation of tears by lacrimal punctal occlusion

Non-infective conjunctivitis

1. Describe the types, clinical picture, complications and treatment of allergic conjunctivitis?

Types:

a. Spring catarrh → Chronic allergic conjunctivitis (type 1 = atopy) hypersensitivity

Etiology:

Due to exogenous antigen ex: UV rays, pollen, dust, Fumes

Symptoms:

- 1- D → Discomfort
D → Scanty whitish ropy mucoid discharge
R → Redness "hyperemia"
- 2- (BLP)
- 3- Itching (severe)
- 4- Mechanical ptosis due to papillae

Signs:

- 1- Palpebral type (Cobble stone papillae):
Large red Flat topped in upper palpebral conjunctive "upper tarsus". Fornix is Free.
They're exposed by eyelid eversion, 1mm covered by sticky discharge full of eosinophils (bulbar or limbal)
- 2- Bulbar type:
Gelatinous masses around the limbus. Usually start at upper limbus. While spot concretions of necrotic epithelium +/- calcium + eosinophils may be seen (Tranta spots)
- 3- Mixed type:
Mixture of palpebral and bulbar type.

Complications:

- 1- Risk of keratoconus
- 2- Punctate micro-erosions (keratitis superficialis of Tobgy####)
- 3- Macro-erosions

- 4- Annular 360 pannus
- 5- Corneal plaque (vernal or shield ulcer)

Treatment:

- a- Symptomatic ttt:
 - dark glasses and cold compresses
 - Vasoconstrictors and antihistaminic drops
 - Systemic antihistaminic may help
 - Avoid exposure to allergens if known
- b- Mast cell stabilizers:
 - Disodium cromoglicate: prevents mast cell degranulation preventing histamine release
 - Lodoxamide
- c- Topical steroids:
 - Only in severe non-responsive cases
 - Prolonged use may cause cataract and secondary glaucoma
- d- Resistant cases:
 - Beta irradiation to the conjunctiva

b. Phlegetynular conjunctivitis → acute allergic conjunctivitis. (type 4= Delayed hypersensitivity)

Etiology:

Hypersensitivity to endogenous antigen ex:

- 1- Staphylococcal (blepharitis)
- 2- Tuberculoptn as in chest T.B
- 3- Streptococcal as in tonsillitis
- 4- Intestinal parasites

Symptom:

- D → Discomfort, burning and foreign body sensation
- D → Watery or mucoid discharge
- R → Redness

- Photophobia and blepharospasm in cases with corneal affection

Sign:

- a- Phlycten:
 - Rounded raised nodule
 - Greyish or yellowish
 - Common at the limbus and bulbar conjunctiva
 - Formed of lymphocytic aggregation covered with intact epithelium, which ulcerates later with 2ry infection
 - Surrounded by a small are of congestion

Complication:

- 1- Corneal phlycten → superficial or deep to bowman's membrane
- 2- Phlyctenular pannus:
 - Any part of limbus
 - Thin and vascular with straight vessels deep to BM
- 3- Ulcers are either limbal or fascicular ulcers.
 - Limbal: (single, multiple or annular)
 - Fascicular is acute serpiginous ulcer that creeps toward the center and supplied by leash of blood vessels
 - On healing its tract leaves an opacity maximum when it stops

Treatment:

- Treat the cause of allergy if possible
- Dark glasses
- Topical steroids for allergy
- Lotions and local antibiotics in cases complicated with mucopurulent conjunctivitis
- Local atropine in cases with corneal involvement
- Fascicular ulcer needs cautery with carbolic acid and actual cautery for blood vessels at the limbus

c. Giant papillary conjunctivitis

Etiology:

Associated with contact lens wear especially extended-wear contact lenses with hard lenses

May result from irritation with ocular prosthesis or by exposed corneal sutures

Symptoms:

- 1- Contact lens intolerance
- 2- Irritation: discomfort, burning and FB sensation
- 3- Watery or mucoid discharge
- 4- Blurred vision from mucus coating of the contact lens
- 5- Hyperemia

Signs:

Giant papillae:

- Affecting mainly superior tarsal conjunctiva
- They're large, flat-topped and covered with mucus

Treatment:

- Discontinuation of use offending lens (symptoms resolve but papillae will persist for several months)
- Topical ttt as in spring catarrh

2. Phlyctenularkeratoconjunctivitis

Discuss etiology, clinical picture and treatment of spring catarrh? (98)

See the answer of the previous question

3. Mention differential diagnosis of Red eye

1. Acute congestive glaucoma
2. Iridocyclitis
3. Corneal ulcer
4. Conjunctivitis
5. Sub conjunctival hemorrhage
6. Episcleritis – scleritis

7. Endo/pan ophthalmitis
8. Orbital cellulitis
9. Cavernous sinus thrombosis

M C Q

1. Regarding pterygium, all are true except:

- a) It's a triangular fibro-vascular tissue with elastoid degeneration of the stromal collagen
- b) Composed of head, neck and body
- c) Simple excision will decrease the incidence of recurrence
- d) Could be a progressive or regressive type

ANSWER: C "Recurrence is common"

2. Dry eye can be caused by all the following except:

- a) Obstruction of the lacrimal puncti
- b) Collagen vascular diseases
- c) Atrophy of the accessory lacrimal glands
- d) Vit. A deficiency

ANSWER: A "Tear deficiency occurs due to obstruction of the ducts of the main lacrimal gland"
 "Obstruction may be at the level of the puncti causing Obstructive Epiphora"

3. The antigen in phlyctenular conjunctivitis could be all except? (2011)

- a) Intestinal parasite
- b) Tuberculoprotein
- c) Staphylococcal blepharitis
- d) Pollens

Answer: **(D)**

4. Papillae of spring catarrh have the following characteristics except? 2012

- a) Large papillae
- b) Flat topped
- c) Always involving the fornix
- d) Ropy discharge

Answer: (C)

5. In spring catarrh the following is true? (2016)

- a) It is type 1 hypersensitivity Reaction
- b) Characterized by follicular formation
- c) The antigen can be intestinal parasite
- d) Steroid eyedrops should be used as 1st line of ttt

Answer: (A)

6. All of the following are non-specific signs in conjunctivitis except :

- a) Subconjunctival hge.
- b) Papillae.
- c) Follicles.
- d) Pseudomembranes

Answer: (C)

7. Corneal damage with trachoma is due to:

- a) Trichiasis
- b) Dryness

- c) Lagophthalmos and exposure
- d) All of the above

Answer: (D)

8. Pre-auricular lymphadenopathy occurs with the following conjunctivitis:

- a) Vernal keratoconjunctivitis.
- b) Phlyctenular keratoconjunctivitis.
- c) Viral conjunctivitis.
- d) Angular conjunctivitis.

Answer: (C)

10.Etiology of pterygium:

- a) Neoplastic
- b) Infection.
- c) Inflammation.
- d) Degenerative.

Answer: (D)

11.Episcleritis is similar to phlycten clinically but differs in being:

- a) Tender.
- b) Flat.
- c) Pigmented.
- d) Multiple.

Answer: (A)

12.The leading cause of preventable blindness worldwide:

- a) Age related macular degeneration.
- b) Glaucoma.
- c) Diabetic retinopathy.
- d) Trachoma.
- e) Senile cataract.

Answer: (D)

The lacrimal system

Anatomy

DISCUSS:

1. The anatomy of lacrimal drainage system. (2004)

1. Gross anatomy:

- a. Two puncti.
- b. Two canaliculi.
- c. Lacrimal sac
- d. Nasolacrimal duct (NLD).

A. Puncti:

- Present on lid margin, near its posterior border.
- At a distance of 6 mm, lateral to medial canthus, on 2 slightly elevated portion called lacrimal papillae.
- Not visible (normally), except if lids are pulled away from eye or in ectropion.

B. Two canaliculi:

- These are fine tubes which carry tears from the puncti to the lacrimal sac.
- Each canaliculus is formed of 2 portions:
 - * Vertical – 2 mm.
 - * Horizontal – 6 mm.
- The 2 canaliculi open into the lacrimal sac by a common canaliculus which opens at the junction between the upper 1/3 & lower 2/3 of the sac.

C. *Lacrimal sac:*

Site: → In the lacrimal fascia in medial orbital wall.

Size: → When distended, it's 8 * 12 mm.

Parts:

- Fundus: upper part, extends above medial palpebral ligament.
- Body: main part.
- Neck: narrow and continuous with NLD.

It's enclosed in the lacrimal fascia.

D. *Nasolacrimal duct:*

- It's 12-16 mm tube, which carries tears from lacrimal sac to the nose.
- It opens in inferior meatus.
- Direction: downwards, slightly backwards & laterally.
- Its opening in the nose is guarded by a valve (Hasner's valve).

2. *Microscopic anatomy:*

- Puncti & canaliculi:** → Lined by stratified squamous epithelium.
- Sac of NLD:** → Lined by 2 layers of columnar epithelium containing goblet cells.
- NLD:** → Surrounded by C.T. rich in veins. Congestion of this plexus leads to nasolacrimal duct obstruction (NLDO).

Disorders of Lacrimal System

SHORT QUESTIONS

1. Causes and investigations of a watery eye

Causes

A. Increased secretions "lacrimation"

Causes:

- a) Emotional conditions
- b) Reflex lacrimation due to corneal ulcer, iritis, foreign body or inflammation

B. Decreased drainage "epiphora"

Causes:

a. diseases of the upper lacrimal passages

1) Lids:

- Ectropion
- Irregular posterior border
- Orbicularis Muscle Paralysis "Pump failure"

2) Punctum:

- Stenosis or obstruction either:
 - a- Congenital
 - b- acquired:
 - post inflammatory scarring due to trachoma, herpes
 - post traumatic fibrosis

3) Canaliculus:

- Stenosis or obstruction either:
 - a- Congenital
 - b- Foreign body e.g. lash
 - c- Post inflammatory and post traumatic scarring
 - d- Canaliculitis:
 - Caused by actinomyces israelii
 - punctum is red and prominent
 - Gritty sensation or probing
 - on pressure a mucopurulent discharge with yellow-black concretions (sulfur granules) may be expressed
- Treated by canaliculotomy and topical antibiotics

b. Diseases of the lower lacrimal passages

1) Lacrimal sac:

- Congenital absence
- Adhesion from repeated inflammation (dacryocystitis; acute or chronic)
- Tumors of lacrimal sac
- Fracture of orbit (medial wall)

2) Naso-lacrimal duct:

- Congenital: imperforation of Hasner's valve
- Acquired:
 - a- Venous congestion
 - b- Post inflammatory stricture
 - c- Tumors of bony canal

3) Nose:

- Polyps or tumors
- Deviated septum
- Hypertrophy of inferior turbinate
- Rhinoscleroma

Investigations

1) Ocular examination:

- a) Exclude causes of lacrimation
- b) Examine lid margin for ectropion, irregularities, punctal obstruction
- c) Examine lacrimal sac area of swelling or signs of acute inflammation

2) Diagnostic tests:

a) Fluorescein dye test:

- Put a drop of fluorescein in conjunctival sac
- after 2 mins ask the patient to blow his nose in a white handkerchief
- if green color: Patent passages
- No green color: Obstruction

b) Regurge test:

- Ask patient to look up and pull the lids laterally to reveal punctum
- press by little finger on the lacrimal sac below the medial palpebral ligament
- "UPWARDS, BACKWARDS and MEDIALY"
- If -veregurge = Normal or Upper canalicular obstruction or Mucocoele or Pyocoele
- If +veregurge = NLD Obstruction

c) Diagnostic Syringing:

- Dilatation of lower canaliculus then syringing with saline is done
- If regurge from other punctum: NLD Obstruction or Common canalicular obstruction
- If regurge from same punctum: Canalicular obstruction

d) Jones-dye test

- Test I: Fluorescein test is done "if no green color"
- Test II: The lacrimal passages are irrigated with saline

- If Fluorescein is recovered: Partial Obstruction or Pump Failure
- If No Saline reach nose: Complete Obstruction

3) Radiological examination:

a) Dacryocystography:

- A radio-opaque dye "Lipidol" is injected in the canaliculus and repeated X-rays are taken in intervals to detect filling and time needed to empty the sac
- If level of the dye is seen: Obstruction
- If rat tail appearance: Stricture
- If Delayed emptying: Failure of lacrimal pump

N.B: Normal emptying takes maximum 15 mins for the sac and the duct 30 mins

b) X-ray on the nose and sinuses for diagnosis of tumors or fractures

2. Enumerate causes of epiphora "Very Very Important"

A. Diseases of the upper lacrimal passages

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 - b- Post inflammatory stricture
 - c- Tumors of bony canal

3) Nose:

- Polyps or tumors
- Deviated septum
- Hypertrophy of inferior turbinate
- Rhinoscleroma

3. Clinical picture and treatment of dry eye

Clinical picture

Symptoms

- Irritation, discomfort, itching and burning
- Foreign body sensation

Signs

- Deficient precorneal tear film
- Punctate epithelial erosion of the cornea
- Watery or mucoid discharge
- Loss of corneal luster in severe cases, corneal opacification with loss of vision occurs

Investigations

- a) Tear film break-up time (BUT):
 - is diminished

- Normally it's 15 mins
- b) Schirmer's test:
 - a normal person wets 10-30 mm of a Whatman number 41 filter paper strip (5 mm wide X 30 mm long) in 5 mins
 - Values less than 5 mm indicate hyposalivation
- c) Rose Bengal Staining of devitalized epithelial cells

Treatment

- a) Treat the cause
- b) Protective glasses and contact lenses
- c) Replacement with artificial tears and lubricants
- d) Preservation of tears by lacrimal punctal occlusion
- e) Systemic steroid.

4. Acute Dacryocystitis "Def, C/P, Complications"

Definition

It's acute suppurative inflammation of the lacrimal sac

Clinical picture

Symptoms

- 1) General:
 - Severe pain
 - Fever
- 2) Local:
 - Painful swelling at the site of the sac
 - epiphora

Signs

- 1) Swelling: red, hot, tender below the medial canthus
- 2) Regurge test: painful and -ve "due to edema of canaliculi"

Complications

- 1) Lacrimal fistula: sac may burst anteriorly through the skin
- 2) Pyoceles: canaliculi may become obstructed

- 3) Orbital cellulitis
- 4) Cavernous sinus thrombosis
- 5) Chronic Dacryocystitis

5. Chronic Dacryocystitis "Def, etiology, C/P, Complications"

Definition

It's chronic inflammation of the lacrimal sac secondary to obstruction of the naso-lacrimal duct

Etiology

- 1) Stasis of tears: in the sac, due to NLD obstruction
- 2) Infection: Pneumococci "80% from nose and sinuses"

Clinical picture

Symptoms

- 1) Watery eye
- 2) Discharge

Signs

- 1) The inner canthus is red and hyperemic
- 2) Swelling of the lacrimal sac below the medial palpebral ligament
- 3) Regurge test is +ve: pressure on the swelling causes regurge of mucous and pus

Complications

- 1) Lid: Epiphora, eczema and ectropion (Vicious circle)
- 2) Conjunctiva: Chronic Conjunctivitis
- 3) Cornea: Hypopyon Ulcer
- 4) Endophthalmitis following intraocular operations
- 5) Mucocele and pyocele: if the canaliculi are obstructed
- 6) Lacrimal sac:
 - Acute Dacryocystitis
 - abscess
 - lacrimal fistula

Treatment

Aim:

- To restore communication between lacrimal sac and the nose
 - To treat infection
- 1- **Treatment of congenital dacryocystitis:**
 - Antibiotics: systemic and topical (drops and ointment)
 - Hydrostatic massage: press on lacrimal sac in a downward direction. This may help remove any remnants for a long period up to 6 month.
 - Probing
 - Irrigation: repeated syringing with saline may cure the condition
 - Dacryocystorhinostomy
 - 2- **Treatment of acquired dacryocystitis:**
 - Treatment of the cause of obstruction
 - **Dacryocystorhinostomy:** Operation of choice
 - Dacryocystectomy: in neglected cases

6. Dacryocystorhinostomy

Principle:

Create a surgical opening between lacrimal sac and nasal mucosa of the middle meatus allowing drainage of tears directly into the nose bypassing the obstructed NLD

Indications:

- 1- Chronic dacryocystitis
- 2- Mucocele of lacrimal sac
- 3- Lacrimal fistula

Contraindications:

- 1- Bad lacrimal sac: extensive adhesions and neglected cases
- 2- Bad nasal mucosa: atrophic rhinitis and polypi
- 3- TB and tumors of the sac
- 4- Hypopyon ulcer

MCQ:

1. The mucin layer of the pre-corneal tear film is secreted by: (6/2016)

- a. Goblet cells.
- b. The lacrimal gland.
- c. The Meibomian gland.
- d. The limbal stem cells.

Answer: (a)

2. The following statements about the pre-corneal tear film are correct, except:

- a. It provides nutrition to the cornea.
- b. Protects the cornea through its anti-bacterial effect.
- c. It lubricates the corneal surface.
- d. It's not affected by deficiency of goblet cells.

Answer: (d)

3. The lacrimal secretory system consists of the following parts, except: (7/2011), (6/2016)

- a. Lacrimal gland.
- b. The accessory lacrimal gland.
- c. The goblet cells.
- d. The lacrimal sac.

Answer: (d)

4. Nasolacrimal duct opens in: (6/2016)

- a. Upper nasal meatus.
- b. Inferior nasal meatus.
- c. Middle nasal meatus.
- d. Nasopharynx.

Answer: (b)

5. Pneumococci can cause:

- a. Acute dacryocystitis.
- b. Chronic dacryocystitis.

- c. Atypical hypopyon ulcer.
- d. Ulcerative blepharitis.

Answer: (b)

6. The first line of TTT in a case of congenital nasolacrimal duct obstruction during the first 4 months of life is:

- a. Massage and antibiotic eye drops.
- b. Probing and syringing.
- c. Dacryocystectomy
- d. Dacryocystorhinostomy.

Answer: (a)

7. The most common Causative organism in a case of chronic dacrocystitis is:

- a. Pneumococci
- b. Staphylococci
- c. Diphtheriabacili
- d. None of the above.

Answer: (a)

8. Flourescein eye drops are used in the following except:

- a. Diagnosis of corneal ulcer.
- b. Diagnosis of diabetic retinopathy.
- c. Applanationtonometry
- d. Lacrimal drainage system evaluation.

Answer: (b)

9. DefectiveLacrimal drainage can result in any of these conditions except:

- a. Chronic dacrystitis
- b. Trichiasis
- c. Stenosis of lacrimalpuncti
- d. Mucocele of lacrimal sac

Answer: (b)

10.Pneumococcus is the commonest organism in chronic dacrocystitis:

- a. True
- b. False

Answer: (a)

11.The orbital portion of lacrimal gland is responsible for:

- a. Reflex tear secretion
- b. Basic tear secretion
- c. Mucin layer secretion
- d. None of the above

Answer: (a)

12.A +veregurge test is diagnostic of:

- a. Nasolacrimal duct obstruction
- b. canalicular obstruction
- c. Punctal occlusion
- d. Ectropion

Answer: (a)

13. Investigation in a case of epiphora include all of the following except:

- a. Examination of the cheeks and face for scars
- b. Examination of the lid margin
- c. Jones dye test
- d. Fluorescein angiography

Answer: (d)

14. Complication of acute dacrocystitis include all of the following except:

- a. Cavernous sinus thrombosis
- b. orbital cellulitis
- c. Fistula
- d. Squint

Answer: (d)

15. A case diagnosed as having chronic dacrocystitis will present with the following clinical picture except:

- a. Fullness related to the medial canthus
- b. Epiphora
- c. Swelling below the medial canthus
- d. -ve regurge test

Answer: (d)

16. Contrast dacryocystography is indicated in the following conditions:

- a. To confirm the site of the obstruction especially prior to lacrimal surgery
- b. to aid in the diagnosis of diverticula and fistula
- c. To diagnose the filling defects caused by tumours or stones
- d. All of the above

Answer: (d)

Cornea

ANSWER THE FOLLOWING QS:

1. Describe anatomy (gross and microscopic) of cornea.

Gross anatomy (Diameter – Thickness – Curvature)

Diameter:

- Adult: V:11mm x H:12mm.
 - Children: V:10 mm x H:11mm.
- {vertical diameter is less than (1mm) because of conj overlapping.}

Thickness

- thinner at center than periphery.
- Centre: 0.5-0.6mm - at limbus: 0.7-1mm. -

Curvature:

- R→7.8mm.

Microscopic picture: (5 layers).

1. **Epithelium(5-6 layers):**

- o str. Squ. Non-keratinized.
- o deep basal layer of columnar cells on basement membrane, their nuclei are oval with long axis perpendicular to BM.
- o 2-3 layers of wing polyhedral cells, their nuclei parallel to surface.
- o 2 rows of flat squ. Non-keratinized epith. Jointed together with tight junctions.
- o Acts as outside guard of cornea /capable of regenerate.

2. **Bowman's layer:**

Superficial acellular condensed layer of stroma elastic incapable of regenerate (heal by scar).

3. **Stroma:**

- Represent 90% of thickness
- About 100 regularly arranged layer of collagen fibers arranged as bundles.
- Closely packed together.
- Run parallel to each other.
- Cross each other perpendicularly.
- bathed in mucopolysaccharide containing few keratocytes.
- Has spaces at periphery contain:
 - fixed cells (keratocytes).
 - wandering cells (macrophage).
- Incapable of regeneration (heal by scar).

4. **Descemet's membrane:**

- Elastic membrane represent basement membrane of endothelium (cellular).
- Capable of regenerate.
- Elastic and resistant

5. **Endothelium:**

- Single layer of flat hexagonal endothelial cells function: Pump out water (deturgescent).
- Count: 4000 cell/mm²keep functioning till 400 cells/mm².
- Incapable of regeneration, when damaged neighbouring cells enlarge(polymegathism and polymorphism).

Anatomy of limbus: لازم يتكتب مع السؤال

- 1) Corneal epithelium continues with conj. Epith.
- 2) Bowman's membrane ends in a round border short distance from limbus.
- 3) Stroma continue with sclera.
- 4) Descemet's membrane end in around border known as (schwalbs line).
- 5) Endothelium continue as endothelium of trabecular meshwork and iris endothelium.

Nutrition: TAL

1. tears(oxygen)
2. aq. Humour(glucose)
3. limbal capillary

N. Supply:

5th cranial nerve (trigeminal) → ophthalmic division → naso ciliary n → long ciliary n → 3 plexuses

- 1) 1.Stromal plexus.
- 2) 2.Sub-epithelial plexus.
- 3) 3.Intraepithelial plexus.

It has the least threshold of pain in the body.

2. Discuss signs, symptoms and complication of CU

Def:

loss of corneal epithelium and superficial layers of stroma.

Symptoms:

- a) Pain sever and stitching or pricking and increase with lid movement.
 - b) blepharospasm.
 - c) lacrimation
 - d) photophobia
- (b,c,d are reflexes for pain{BLPH})
- e) decrease acuity of vision due to:
1. Opaque area of cornea (central).
 2. Associated uveitis.
- f) Red eye.
 - g) Special features:
 - o The onset of pain and photophobia in a patient with conjunctivitis may denote the formation of a corneal ulcer.
 - o Pain is characteristically absent in neuroparalytic keratitis.

- o Severe neuralgic pain before the appearance of any signs is suggestive of herpes zoster.

Signs:

- o lid: Edema (due to blepharospasm)
- o Conj.: Ciliary injection and edema (chemosis)
- o Cornea:
 - grey area of cellular infiltration in the bed and edges of the epithelial defect without stromal loss.
 - loss of luster (loss of intact epithelium).
 - positive fluorescein test (diagnostic test), the area of the ulcer devoid of epithelium is stained giving a green glow of the ulcerated area in blue light.
 - Rose Bengal 1% stains the diseased epithelium.
 - Anterior chamber examination shows flare, cells and even hypopyon.

Morphology:

- bacterial ulcers usually rounded or oval
 - HS ulcers dendritic in shape
 - Typical hypopyon are serpiginous
- o Grading:

<i>Risk</i>	<i>Size</i>	<i>Depth</i>	<i>Scleral involvement</i>
Mild	< 2mm	< 20%	Not involved
Moderate	2-5mm	20-50 %	Not involved
Severe	> 5mm	> 50%	affected

Complication: VVVV imp

a) Non-perforated

1. *2ry severe iridocyclitis* associated with hypopyon formation:
 - due to toxin diffusion.
 - common with pneumococci and pseudomonas infection
 - post synechia may occur
 - hypopyon is sterile unless the cornea is perforated.

2. *2ry glaucoma:*

due to plasmoid aqu. Or hypopyon, progressive or Deeping of the ulcer with stromal thinning predisposing by:

- Steroid therapy
- Low immunity as diabetes
- Virulent organism as B strep and pseudomonas

3. *Descematocele:*

- herniation of Descemet's membrane at ulcer base due to high IOP after loss of epithelium and stroma as a clear vesicle at the ulcer base.
- Not common in children and hypopyon ulcers due to weak DM which leads to rapid perforation.
- either rupture or heal

4. *Complication of healing (scarring +/- vascularization)*

i. *Typical opacities:*

- superficial → Nebula (faint iris and details are visible)
- Deep → Leucoma (dense: no visible iris details)

ii. *Atypical opacities:*

- facet:
depressed corneal scar lined by epithelium (not stained by fluorescein but pooling of the stain)
- kerato-ectasia:
ectasia (out bulging of weak corneal scar)
- pseudopterygium:
adhesion of conj. Fold to ulcer base

b) perforated ulcer: causes severe pain relieved by gush of fluid

1) *Small Peripheral:* leucoma adherent:

- o Pear shaped pupil
- o Irregular depth of AC
- o Leucoma +/- Pigments
- o If so peripheral: localized PAS

2) *Small center perforation:*

- *Leucoma non-adherent* or
- *Corneal Fistula:*
 - Due to dislodgement of the plug formed by the fixed keratocytes with secondary epithelization of the perforation track
 - Diagnosed by +ve river sign (siedle test)
- PAS due to prolonged loss of AC causing secondary closed angle glaucoma if healed.
- Acquired anterior polar cataract.

3) Large corneal perforation leads to iris prolapse and PAS with anterior staphyloma ending with a blind painful eye

Complication of perforation (infection- hypotony-anterior polar cataract)

4) *large central ulcers:*

- Infection - Endophthalmitis/panophthalmities
- Expulsive Hge (due to sudden decrease IOP)
- Sub laxation and dislocation of lens

3. Describe clinical picture of various types of hypopyon CU, its complication and treatment

Types:

- 1) ***Atypical hypopyon ulcer 20%*** caused by organism other than pneumococci in dacrocystitis (clinical picture previously describes).
- 2) ***Typical hypopyon ulcer 80%*** (has the same clinical picture but):
 - More severe Iridocyclitis that may show hypopyon
 - Central
 - With an undermined central advancing creeping edge and sloping peripheral healing vascularized edge (serpiginous)
 - Associated with posterior corneal abscess.
 - more severe secondary glaucoma

- descemetocoele is rare due to post abscess formation (weak cornea liable to perforate)
- perforation is more common
 - o post abscess formation
 - o spread toward centre which is thinner
- associated with dacryocystitis

Lines of treatment

a) Topical treatment:

- 1- Broad spectrum antibiotics E. D and E. Clint
 - 2- Cycloplegic ED
 - 3- Eye patching except with discharge
 - 4- Hot fomentation
- 1- Topical antibiotics:
- Either monotherapy broad spectrum (fluoro-Quinolones)
 - 3rd generation e.g.: 3% ciprofloxacin ED
 - 4th generation e.g.: moxifloxacin 5%ED
 - Or Fortified ED: fortified amino glycoside (against gram) garamycin or tobramycin 15%mg/dl –amitracin
 - And fortified cephalosporins—vancomycin 50%mg/dl
- Dose: loading every 5 min for 30 min
- Maintain: every 30 min for 24 h then 5 times daily
- 2- Cycloplegic ED: Atropine ED
- In children atropine E. oint
 - In atropine sensitivity: cyclopentolate 1%
 - In pupil resist give sub. Conj. Injection of myclricain
 - Atropine 1%
 - Adrenaline 0.1%
 - Novocaine 2%
- 3- Patching: for avoiding blinking and photophobia

CL: in conjunctivitis with discharge

- 4- Bandage soft cl.: collagen shields impregnated with antibiotic.

b) Systemic:

- 1- Sys. Antibiotic effective in
 - *ulcers near limbus
 - *scleral involvement
 - *perforated
- 2- NSAIDs (never cortisone) –anti-inflammatory.

c) Surgical treatment

- 1) Cauterization by carbolic acid
- 2) Paracentesis: aspiration of aqueous and hypopyon
- 3) Conj. Flap: avoid perforation and decrease complication
- 4) Therapeutic CL: prevent perforation-
- 5) Tarsorrhaphy:
 - lateral (in lagophthalmous)-
 - Median (in loss of sensation)
- 6) -Therapeutic treratoplasty: last line of treatment.

Complication of treatment

- 1- Iritis:
never use steroids, use NSAIDs*
- 2- 2ry glaucoma:
medical ttt*
- 3- Descemetocoele:
hospitalization+ surgical ttt*
- 4- Perforation:
hospitalization + surgical ttt*
- 5- *Fistula:
destruction of epithelial tract by hot platinum needle.

4. Forms of herpes simplex Keratitis with clinical picture and treatment.

Caused by:

75% HSV type I

25% HSV type II

a) Primary HSV:

- Presenting from 6 months – 5 years.
- Most commonly in the mucocutaneous distribution of the trigeminal nerve including eye.
- Widespread (No antibodies) superficial (Epitheliotrophic virus) lesions.
- *Clinical picture:*
 - 1- Superficial punctate keratitis
 - 2- Superficial punctate erosion.
 - 3- Follicular conjunctivitis.
 - 4- Lid vesicles.
 - 5- NO Stromal lesion as it is an Epitheliotrophic virus and no antibodies.

b) Recurrent HSV:

- After primary infection in any of the three branches of trigeminal nerve. The virus spreads along the axons of sensory nerve endings to the cell bodies in the trigeminal ganglion where inter-neural spread could happen.
(Recurrent HSV keratitis can occur without primary ocular infection)
- It stays in a latent (Dormant) stage, re-activated by:
 - 1- UV exposure
 - 2- Decrease of immunity >> Disease, Steroids, Menstrual cycle, Fever, Stress, Fatigue

Symptoms similar to bacterial keratitis but less severe

• *Clinical Picture:*

- 1- Anterior Uveitis (Hyphema, Hypopyon) with Trabeculitis and secondary glaucoma.
- 2- Interstitial Keratitis:
 - A) Disciform keratitis:** Deep stromal circular central infiltration due to antigen-antibody reaction heals with NO scar.
 - B) Deep necrotizing keratitis:** Actual stromal invasion. Heals with scars and vascularization
- 3- Corneal ulcers:

a) Dendritic Ulcer: Classical corneal lesion in recurrent HSV keratitis.

- Localized corneal lesion: Superficial, linear, branching, central, ends into knobs.
- Bed of ulcer is stained with Fluorescein.
- Epithelial margin is stained with Rose-Bengal.
- No vascularization, No perforation.
- If BM and stroma are not affected healed with NO scar
- Recurrent.
- Accompanied by hypoesthesia.

b) Limbal Ulcer.

c) Geographic (Amoeboid) Ulcer: Enlargement of dendritic ulcer in patients with low immunity OR on topical steroids

d) Deepening of Ulcer: Low immunity, Steroids, Secondary bacterial infection.

e) Meta-herpetic Ulcer: Is a non-infected post-herpes ulcer due to loss of corneal epithelial.

- *Treatment:*

a) Anti-viral Drugs:

Interfere with viral and human DNA synthesis “Toxic”

Cannot be used more than 2 weeks **except Acyclovir**

- Local:
 1. Acyclovir: 3% ointment, 5 times daily up to 60 days least toxic.
 2. Vidarabine: 3% ointment 5 times daily .
 3. Trifluorothymidine (T3F): 1% Eye drops 5 times daily OR every 2 hours for 2 weeks.
 4. Idoxuridine (IDU): 0.1% drops OR 0.5% ointment, Toxic, Resistance developed
- Systemic: for immunosuppressed
 1. Acyclovir: 800 mg tablet 3 times per day for 3 weeks
 2. Valacyclovir (pro-drug)

b) Surgery:

- Debridement of infected epithelium and Topical antiviral drugs:
 - ★ Indicated when patient is non compliant for antiviral, allergic to antiviral or if the

antiviral is not available.

- ★ Contraindicated in case of amoeboid ulcer.
- ★ Done by wiping the corneal surface with a sterile cotton-tipped bud 2mm beyond the ulcer edge.
- Penetrating Keratoplasty in case of opacities.

c) The usual ttt of corneal ulcer : Antibiotic E.D + Atropine + Patching.

5. Forms of Herpes simplex corneal ulcer and treatment

6. Dendritic corneal ulcer/Classical corneal lesion in recurrent HSV

See Above

NOTES (ORAL)

- *Dendritic ulcer is linear and branching*
because it affects corneal epithelium along the pathway of sensory nerve endings. Since the virus is dormant in the trigeminal ganglion and reaches cornea after activated via nerves. The cells infected are called vesicles as they are abnormal with viral DNA and inclusion bodies.
- *The ocular manifestation following exposure to UV light:*
 - 1- Allergic Conjunctivitis (Spring Catarrh)
 - 2- Herpetic Ulcer
 - 3- Pterygium – Pinguecula
- *Pain is much less in viral corneal ulcer than in bacterial*
because the virus is Epitheliotropic affecting epithelium only with superficial ulcer. And inflammation causes Limbal B.V to dilate and release exudate that coats the nerve endings within the ulcer (Hypoesthesia).

GIVE AN ACCOUNT ON:

1. Keratoconus (Outline the management of keratoconus)

Definition:

It's progressive central and paracentral stromal thinning leading to corneal ectasia and apical protrusion due to weakness of corneal collagen.

Characters:

- 1- Bilateral (85%)
- 2- Asymmetrical
- 3- Start around puberty (10-20 years)
- 4- Progress for few years (variable)
- 5- Common association are:
 - a) Systemic diseases: Down's syndrome, Marfan's syndrome
 - b) Local eye disease: Spring catarrh, Retinitis pigmentosa, Contact lens wearers

Clinical Pictures:

a) Symptoms:

Rapid progressive decrease V/A and frequent changes of glasses

Explanation: progressive myopia and astigmatism

b) Signs:

a) Mild keratoconus

1. **External** and **corneal** signs are absent or minimal.
2. **History of:** multiple inadequate glasses corrections of one or both eyes may be noted and may include oblique astigmatism on refraction as well as moderate to high myopia.
3. **Keratometry**
Irregular astigmatic keratometric values.
4. **Placido disc**
Irregular circles with central crowding.
5. **Corneal topography**
Diagnosis can be confirmed

b) Moderate keratoconus

1. **Gross sign:**

- a. Munson sign “on looking downwards causing angulation of lower eyelids”.
- b. Deep AC

2. **Slit lamp signs:**

- a) Appearance of corneal nerves is noted.
- b) Vogt striae “Fine stress line”: In the deep stroma
- c) Corneal scars “superficial or deep”
- d) Deposition of iron in the basal epithelial cells in ring shape at the base of the conical protrusion called the **Fleischer ring**

3. **Keratometry:**

typically increases to 45-52 D

4. **Retinoscopy:**

Distortion of red pupillary reflex may allow observation of “scissoring” or an inferior distortion named the oil drop sign.

c) **Advanced keratoconus**

1. **Slit lamp:**

Vogt striae in 60% of eyes and ulcers and scarring in 70% of eyes.

2. **Keratometry:**

Greater than 52D and enhancement of all corneal signs, symptoms and visual loss or distortion.

3. **Complications:**

acute corneal hydrops

- Continuous stretch and ectasia of the cornea → leads to break in Descemet membrane → acute corneal edema and opacification.
- Resolves in few weeks leaving a residual central opacity.

Management *يجي سؤال لوحده*

a) **Spectacles:**

can be used in early cases before astigmatism becomes irregular.

b) **Rigid contact lens:**

may help in irregular astigmatism but are useless in case of opacity or steep cones.

c) **Surgical procedures.**

1) **Cross linking:**

- Elevate flap “epithelium+ bowman’s”
- Apply riboflavin drops
- Expose cornea to U.V. rays
- Result → this will make collagen layer more compact and prevent progression.

2) *Intra-stromal corneal rings:*

For patients who become intolerant to contact lens “More successful in modest than in advanced cases”

3) *Lamellar keratoplasty:*

Especially deep anterior lamellar Keratoplasty “DLAK”

4) *Penetrating keratoplasty “PKP”:*

Very successful in keratoconus resulting in clear visual axis in more than 90% of cases.

MCQ

1. Corneal transplantation is due to the following except?

- b) Compact arrangement of corneal fibers
- c) Lack of blood vessels
- d) Myelinated nerve fibers
- e) Non-keratinized str. Squ. Epith.

Answer: (C) Myelinated nerve fibers

2. The Following about dendritic ulcer is true

- a) IT's always bilateral
- b) It's usually recurrent
- c) It's usually associated with thick hypopyon

Answer: (C) It's usually recurrent

3. The following are signs of keratoconus except:

- a) Vogt striae
- b) Munson sign
- c) Kayser Fleisher ring
- d) Oil droplet sign on direct ophthalmoscopy

Answer: (C) Kayser Fleisher ring

4. In keratoconus:

- a) Munsons sign is present
- b) Corneal opacity may be seen
- c) Corneal topograghy is diagnostic
- d) all of the above

Answer: (d) all of the above

5. Treatment of simple corneal ulcer should not include

- a) Antibiotic ED -
- b) Antibiotic Oint-
- c) Pilocarpine ED -
- d) Atropine E.oint-
- e) Bandage

Answer: (C) Pilocarpine ED

6. In Leucoma adherent the anterior chamber is:

- a) Shallow
- b) Deep
- c) Irregular
- d) Lost

Answer: (C) Irregular

7. Keratoconus is diagnosed by:

- a) Fluorescein test
- b) Auto refractometer
- c) US
- d) Placido disc

Answer: (D) Placido disc

8. causes of visual impairment in keratoconus include all of the following except:

- a) Myopia
- b) Corneal opacity
- c) Hyperopia
- d) Irregular astigmatism

Answer: (C) Hyperopia

9. the following is true about the antiviral drugs:

- a) they are all applied as night time ointment
- b) they inhibit RNA replication
- c) They can be used for 2 weeks only
- d) Acyclovir is the most potent drug

Answer: (d) Acyclovir is the most potent drug

10. Arcus senilis is characterized by

- a) It's formed of calcium deposits
- b) It starts in the temporal and nasal corneal periphery and spreads circumferentially
- c) It is formed of lipid deposits
- d) It becomes undistinguished from the limbus by the age of 60.

Answer: (C) It is formed of lipid deposits

11. the cornea is covered by

- a) Keratinized str. Squ. Epith.-

- b) Non-keratinized str. Squ. epith.-
- c) -Str. Columnar
- d) Pseudostratified

Answer: (b) Non-keratinized str. Squ. epith.

12. Descemetocoele means:

- a) Inflammation of deep layer of cornea-
- b) Bulging of descemet's membrane due to melting of the overlying corneal layer
- c) Permeant opacity of cornea

Answer: (B) Bulging of descemet's membrane due to melting of the overlying corneal layer

13. Predisposing factors for corneal ulcers include all the following except?

- a) Trauma-
- b) Exposure-
- c) Loss of sensation-
- d) High myopia

Answer: (d) High myopia

14. One of the following is not a complication of corneal perforation

- a) Anterior staphyloma
- b) Posterior staphyloma
- c) Corneal fistula

Answer: (b) Posterior staphyloma

15. The following is true regarding management of bacterial CU

- a) -Fortifier antibiotics should be given hourly and decrease according to response
- b) Steroids starts within 48h to decrease edema-
- c) Sys. Antibiotics should be given iv every 12 h for first 3 days

Answer: (C) Sys. Antibiotics should be given iv every 12 h for first 3 days

16. Double staining pattern of the cornea is characteristic for:

- a) Fungal corneal ulcer
- b) Herpetic corneal ulcer
- c) Exposure keratopathy
- d) Acanthamoebic corneal ulcer

Answer: (B)

17. Infective corneal ulcer include, except:

- a) Bacterial ulcer
- b) Fungal ulcer
- c) Mooren's ulcer
- d) Viral ulcer

Answer: (C)

18. In treating bacterial corneal ulcer, all except:

- a) Antibiotics drops
- b) Vitamin A,C
- c) Mydriatics and cycloplegics drops
- d) Corticosteroids drops.

Answer: (D)

19. Corticosteroids is given in :

- a) Bacterial corneal ulcer
- b) Herpetic corneal ulcer

- c) Fasicularphlyctenular ulcer
- d) Stromal fungal keratitis

Answer: (C)

20. Blood staining of the cornea is due to :

- a) Hyphema
- b) Hyphema with rise in IOP
- c) Corneal edema
- d) Corneal FB

Answer: (B)

21. Munson's sign in:

- a) Corneal fistula
- b) Corneal dystrophy
- c) Keratoconus
- d) Corneal facet

Answer: (C)

22. In corneal edema all except:

- a) There is increase in corneal diameter
- b) There is increase in corneal thickness
- c) Cloudy cornea
- d) Predispose to corneal vasculatization

Answer: (A)

23. Topical steroids are contraindicated except in:

- a) Phlectenularfasicular ulcer.
- b) Dendritic ulcer.
- c) Typical hypopyon ulcer.
- d) Atypical hypopyon ulcer.

Answer: (A)

24.Staphylococci can cause:

- a) Acute dacryocystitis.
- b) Sty.
- c) Atypical hypopyon ulcer.
- d) Ulcerative blepharitis.
- e) All of the above.

Answer: (E)

Sclera

GIVE A SHORT ACCOUNT ON

1. staphyloma:

Definition:

Ectasia of the outer coat lined by atrophic middle +/- inner coat.

Types:

- **Anterior:**
 - **Etiology:** pathological/ perforated large corneal ulcer.
 - **Site of weakness:** Central (total) or peripheral (partial).

- **Middle coat:** Iris
- **Outer coat:** Pseudo cornea (epithelium & scar tissue).

- **Inter-calary:**
 - **Etiology:** Absolute glaucoma.
 - **Site of weakness:** Schlem's canal e.g. bad repair or wound.
 - **Middle coat:** Root of iris.
 - **Outer coat:** Limbus

- **Ciliary:**
 - **Etiology:** Absolute glaucoma.
 - **Site of weakness:** ant, ciliary vessels & nerves (4 mm from limbus).
 - **Middle coat:** Ciliary body
 - **Outer coat:** Sclera

- **Equatorial:**
 - **Etiology:** Absolute glaucoma.
 - **Site of weakness:** Vortex.
 - **Middle coat:** choroid +/- Retina
 - **Outer coat:** Sclera.

- **Posterior:**
 - **Etiology:** High pathological myopia.
 - **Site of weakness:** lamina cribrosa & posterior ciliary vessels.
 - **Middle coat:** Choroid +/- Retina
 - **Outer coat:** sclera.

N.B:

Posterior: - care during local anaesthesia.

-Diagnosed by fundus & B-US.

Total staphyloma: buphthalmous.

Treatment:

All of them are blind painful ugly eye except post. Staphyloma.....so :

- **Not markedly bulging:**

Cover the eye with contact shell.

- **If markedly bulging:**

Enucleation or evisceration.

D.D for blue thin sclera:

- Neonate
- Buphthalmous – high axial myopia
- Osteogenesis imperfecta
- Over scleritis

DIFFERENTIATE BETWEEN:

	<i>Episcleritis</i>	<i>Anterior scleritis</i>
Etiology:	1. Rheumatoid arthritis (50%) common in middle aged female 2. Infective e.g, HZO 3. Idiopathic.	
Symptoms:	Tenderness -Discomfort-lacrimation-redness.	1. Pain+Blepharospasm-Lacrimation-Photophobia 2. Redness- tenderness 3. Drop of vision
Signs (type):	Nodular/Diffuse	1. Nodular 2. Diffuse 3. Necrotizing (may lead to perforation)
Complications:		1. Uveitis 2. Keratitis 3. Staphyloma
Treatment:	1. Of Cause 2. NSAID 3. SAID (short course topical) D.D: phlycten in	1. Of the cause 2. Topical steroids +/- systemic steroids & immunosuppressive 3. Atropine if uveitis or keratitis

	nodular type.	
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M C Q

1. The only staphyloma with normal IOP is:

- a) Partial anterior staphyloma.
- b) Ciliary staphyloma.
- c) Intercalary staphyloma.
- d) Equatorial staphyloma.
- e) Posterior staphyloma.

ANSWER: (E)

2. Ciliary injection in all except:

- a) AACG.
- b) Corneal ulcer.
- c) Anterior uveitis.
- d) Episcleritis.

ANSWER: (D)

Lens

ANSWER THE FOLLOWING QUESTIONS:

1. Describe the anatomy of the lens.

(Surface ectoderm)(Highest ptn content in the body)

Macro anatomy:

1. Characters:elastic,transparent and Avascular.

2. Shape:assymetrical biconvex lens more convex posteriorly.

3. Radii of curvature:

Anterior surface:10mm

Posterior surface:6mm

4. Site: (patellar fossa) bet. The iris and the anterior vitreous separated from it by the retrolental space of berger containing the aqueous humor ,the lens is suspended by the zonule of zinn bet. It'sequater and the ciliary processes

5. Colour:

Young age: Transparent (all rays are refracted)..

With age: becomes grayish yellow(sclerosis).....(some rays are reflected).

6. Poles: anterior and Posterior poles.

7. Axis: line joining the two poles.(4.5mm)

8. Equater: junction between anterior and posterior surfaces.(equatorial diameter=9-10mm)

Micro anatomy:

1. Capsule: highly elastic and semipermeable membrane secreted by the anterior subcapsular epithelium(A cells) with the thickest part anteriorly(24um) and the thinnest at the posterior pole(4um).

2. Subcapsular epithelium: one layer of cubical cells that elongate towards the equator. (there is no posterior cells).

3. Lensfibures: Elongation of the equatorial cells which is divided into:

A) cortex: peripheral,new and soft (RI=1.39).

B) Nucleus: central,old and hard (RI=1.42).

The lens contains **Sutures** which are the site of articulation of fibers (**Y-Shaped**, Erect

anteriorly and inverted posteriorly).

Function:

1. Second eye refractive medium.
2. UVfilter: protect the retina.
3. Accommodation: due to changing of curvature by contraction of the circular muscle and relaxation of the zonular fibers.

Refractive power: is +18 inside the eye and in the unaccommodated state.

2. Enumerate various forms of congenital cataract. (2003)

- Anterior polar cataract.
- Posterior polar cataract.
- Lamellar cataract.
- Rubella Total cataract. (ودول المهمين)
- Nuclear cataract.
- Blue dot cataract
- Sutural cataract: Y-Shaped.
- Coronary cataract
- May be combined.

3. Discuss congenital cataract.

- **Definition:**
Lens opacification discovered at birth or shortly after birth.
- **Etiology:**
 - Hereditary.(MC)
 - Maternal or fetal hypocalcemia.
 - Malnutrition.
 - Maternal hypoxia

- Metabolic “Galactosemia”
- Infection “Intrauterine” as rubella.
- Irradiation.
- Teratogenic drug
- **Characteristics:**
 - Commonly bilateral.
 - Commonly associated with other congenital anomalies.
 - Commonly stationary course “No progression” except Rubella.
 - Soft cataract (soft lens)
- **Types:**
 - a) Anterior polar cataract:**
 - Aetiology:
Delayed formation of A.C, Prolonged contact between the cornea and lens which leads to Irritation of sub capsular epithelium at the anterior pole → its proliferation and formation of mass of cells + calcium deposition → opacity +/- persistent pupillary membrane(remnants of the tunica vascularis lentis).
 - Shape: could be pyramidal or re-duplicated or hourglass.
 - **N.B:** Congenital anterior polar cataract characterized by -ve history of red eye.
 - b) Posterior polar cataract.**
 - Aetiology:
persistence of remnants of the hyaloid artery (Mittendorf dot).
 - Shape:
Disc shaped opacity at the posterior pole.
 - Vision:
Decreases markedly “As the opacity is close to the nodal point”
 - c) Lamellar cataract (Zonular cataract)**
 - **Def:**
Lens opacification involving one or more lamellae of the lens.
 - **Aetiology:**
 - Hereditary
 - Malnutrition of the mother during pregnancy.

- Low vit D and calcium needed by pyramidal cells as the child often shows evidence of rickets and abnormality of permanent teeth.
- *Shape:*
with the pupil dilated, the Lens opacities composed of:
 - Central disc → Explanation:
At vitamin D and calcium deficiency → the pyramidal cells form opaque fibers → after recovery by taking calcium the pyramidal cells form new clear fibers which push the opaque fibers inward → opaque lamella
 - Projection → Explanation:
The equatorial cells don't recover at the same time so some of them recover forming clear fibers those beside them continue to form fibers opaque. This resembles **cart wheel** appearance.

d) Rubella Total cataract:

Rubella infection to the mother in the 1st trimester. The cataract operation is risky in this patient due to associated cardiac anomalies during general anaesthesia.

- The virus remains dormant inside the lens for 2-3 years
(i.e. operation → endophthalmitis or viral uveitis) so operation must be postponed.
- It starts as a dense pearly white nuclear then progresses to be total.

e) Nuclear cataract:

Confined to the embryonic or fetal nuclei (a central oil droplet cataract is characteristic of galactesmia).

- *Clinical picture: (for all congenital)*
 - *Symptoms:* Given by the mother:
 - a) White pupil "leucoria"
 - b) Defective vision
 - *Signs:*
 - *one of the previous types is seen.*
 - *Assessment of visual acuity*
 - *Fundus should be carefully examined to exclude retinal anomalies*
 - *Full pediatric examination for systemic associations as in case of rubella.*

- *Complications:*

- Bilateral → Nystagmus
- If unilateral → Amblyopia and squint.

- *Treatment: (for all congenital)*

- If unilateral cataract operate as early as possible <6 years to avoid amblyopia then squint.
- Bilateral unequal → bilateral cataract extract < 6 years to avoid amblyopia then squint.
- Bilateral equal → dense cataract + marked decrease in V/A do cataract extraction as early as possible <2 months.
- Bilateral equal → Cataract not dense + decrease in V/A) wait till school age. (Examine visual acuity by landolt's chart).
 - If V/A = 6/18 or better, with glasses or mydriasis then no surgery is needed.
 - If V/A is less than 6/18 → Cataract extraction.

- *Lines of treatment:*

Surgical – Correction of a phakia- Correction of amblyopia

1. Surgical:

a) Irrigation-aspiration:

- Through 2 mm limbal incision (anterior capsulotomy)
- Followed by aspiration of lens matter with simultaneous irrigation with saline using double way cannula.
- The central part of the posterior capsule should be removed by anterior vitrectomy as the infantile posterior capsule mostly opacifies.

b) Lensectomy:

- Removal of the lens using vitrectomy machine.
- Advantage: No posterior capsule opacification
- Disadvantages: R.D

2. Correction of aphakia: To avoid amblyopia by:

- Glasses: (2 pairs, heavy, constricted field)

- Contact lens: (difficult insertion and removal , Complications: Corneal ulcer)
- I.O.L → Power changes with age this explains why 6/18 with accommodation is better than 6/6 without it.

3. ***Correction of amblyopia:***

- Based on forcing the patient to depend on the amblyopic eye for vision.
- Occlusion of preferred eye is carried out, it is important to diagnose amblyopia as early as possible <6 years. Otherwise occlusion will not improve visual acuity.
- The regimen fulltime or part time occlusion depends on the age of the patient and the density of amblyopia.
- The rule of thumb is occlusion for one week per each year of life then monitor the visual acuity in both eyes to avoid inducing amblyopia of the sound eye.

4. **Discuss senile cataract:**

Definition: progressive lens opacity affecting old people in the absence of any other diseases or drugs intake.

Etiology: the exact etiology is unknown, however it may be due to metabolic causes or a decrease in antioxidants or prolonged exposure to UV rays.

Finally undergoes:

- 1) Hydration
- 2) Coagulation of lens ptns leading to opacification of the fibers.

Characters:

- Bilateral usually assymetrical
- Progressive
- Hard
- No associated diseases or drug intake

Clinical picture:

Symptoms:

- White pupil
- Decreased vision (gradual progressive and **painless**)
Unless secondary glaucoma: Rapid **Painful** drop of VA.
- According to the type .

Signs: (morphological types)

- Subcapsular
- Cortical
- Nuclear
- Cortico-nuclear

Types:

Senile cortical cataract:

<i>Stages:</i>	Symptoms	colour	Iris Shadow	Slit Lamp	Red reflex
Incipient	- Halos - Fixed musca - Uniocular diplopia - Hypermetropic shift - Irregular astigmatism - Night blindness	Grayish White	+ve Clear lens fibers bet, the iris and the opacity	- Starts as water vacuoles - Sectorial peripheral deep opacities	Opacities seen as black shadows against red background(the last fibers to be affected are the anterior subcapsular)
Immature	Gradual drop of vision(6/9 to CF)	Grayish White	+ve Clear lens fibers bet, the iris and the opacity	- Starts as water vacuoles - Sectorial peripheral deep opacities	Opacities seen as black shadows against red background(the last fibers to be affected are the anterior

					subcapsular)
Mature	VA: HM	White	-ve	Lens is totally opaque	No RR
Hypermature (lens loses water and shrinks)	VA: HM	White	+ve Clear aqueous bet. The iris and the opacity	- AC:Deep - Iris:tremulous - Capsule:thickened wrinkled with Ca^{++} and cholesterol deposits	No RR

Senile nuclear cataract:

Pathogenesis:

Exaggeration of the physiological sclerosis of the nucleus with age and loss of transparency of the lens.

Symptoms:

- Day blindness
- Myopic shift due to increase in the RI of the nucleus with increase in the total refractive power of the lens.(decrease in the far vision and improvement of the near vision so the patient can read without the reading glasses)

Color:

Grades from light yellow to black (cataract nigra)

Iris Shadow: +ve

Red reflex: dark central disc against a red background.

5. How to prepare and treat a case of senile cataract?

Treatment →surgical removal of the cataract.

Types of operation:

- Extracapsular cataract extraction
- Intracapsular cataract extraction

c) Phacoemulsification

A. *Extracapsular cataract extraction:*

Circular area of anterior capsule of lens is removed, the nucleus is then removed and the cortical lens matter is aspirated leaving the posterior capsule behind to support an intraocular lens.

Methods of hard nucleus removal:

- ➔ Delivery through a large section (6 to 8 mm) at the cornea or at the limbus.
- ➔ Phacoemulsification: The hard nucleus is emulsified using a special ultrasound machine. The nucleus is then aspirated. The procedure is done through a 3mm limbal or corneal incision
- ➔ Laser emulsification: using an erbium-YAG laser through a 2mm incision if the nucleus isn't very hard.

B. *Intracapsular cataract extraction (not used now)*

The lens is removed with the capsule after zonulysis. As there is no posterior capsule left to support a posterior chamber lens, an anterior chamber lens can be implanted.

C. *Phacoemulsification: (the standard of cataract surgery today)*

The lens is emulsified using ultrasound; the idea is to use only a small incision (2.2mm-3.2mm) to enter the eye, thus reducing postoperative astigmatism and patient rehabilitation in these surgeries. Foldable intraocular lenses are used to maintain the small incision.

N.B after removing the lens leaves the patient with a high hypermetropic error of refraction, to compensate for the error, IOL is used.

1. After ECCE ➔ use posterior chamber IOL but a few years after implantation or no implantation. The remaining posterior capsule may opacify, the opacified capsule may be opened using YAG laser (laser capsulotomy).
2. After ICCE ➔ Anterior chamber IOL.
3. After phacoemulsification ➔ Foldable IOL

Times of operation:

- If phacotechnique is decided, the earlier the surgery the better.
- If ECCE technique is decided, the surgery is carried out when the patient is unable to do his work irrespective of the stage.
- Start the surgery with the more advanced eye except if the is intumescent cataract.

6. Definition, Causes, Clinical picture and Management of complicated or secondary cataract (2000, 2007, 2008, 2009). V.V.IMP

Definition:

Opacity of lens due to local ocular disease, systemic disease or drug induced

Etiology:

- *Local ocular causes:*

1. Chronic iridocyclitis/Chorio-Retinitis.
2. Absolute glaucoma.
3. Acute congestive glaucoma “glaucomoflecken”(anterior subcapsular).
4. Retinitis pigmentosa.
5. Intra-ocular tumors: Melanoma, Retinoblastoma.
6. High axial degenerative myopia.
7. Central corneal perforation(Acquired anterior polar)
8. Long standing Retinal Detachment
9. Ectopia lentis..

- *Systemic causes:*

A) Endocrinal:

- DM (Pre-senile as senile but at a younger age – True cataract/snow flake)
- Hypo-parathyroidism
- Cretinism

B) Metabolic

- Galactosemia

C) Irradiation

- Infra-Red, X-Rays, Microwaves

D) Heavy ankylostoma infestation

- *Drug induced:*

- 1- Topical **steroids**, Adrenaline, Pilocarpine
- 2- Systemic **steroids**, Amiodarone, Ergots

3- Toxic >> Naphthalene, Thallium

Clinical Picture:

- 1- Posterior subcapsular: Due to absence of epithelium and thin capsule.
- 2- Progressive.
- 3- Poly chromatic luster: Due to diffraction of slit lamp light by deposition of cholesterol crystals.
- 4- Evidence of the cause: Posterior synachiae, KPs, LPs.
- 5- Disproportionate drop of vision to density of cataract.

Management:

Surgery depends on the age.

1. Soft cataract removed by irrigation aspiration method, Pars-Plana lensectomy.
2. Hard cataract managed exactly like senile cataract
 - *Extra-capsular cataract extraction, *Intra-capsular cataract extraction,
 - *Phacoemulsification

N.B: It is important to make sure that drop of VA is mainly due to cataract only by good preoperative examination and retinal function.

N.B ttt depends on the cause.

Prognosis is not good.

- ❖ **In case of uveitis** it's better to wait for 3-6 months.
- ❖ **In case of diabetics** there is a risk of infection, Hge and delayed wound healing
- ❖ **In case of glaucoma** the two operations may be done in two separate occasions with glaucoma surgry first then cataract after 1-3 moths or they could be combined.
- ❖ **In case of retinal detachment** ; Do ECCE for visualization of retinal condition.
- ❖ **Control the general disease 1st then do cataract surgery.**

7. Discuss traumatic cataract:

- *Definition:*

Any lens opacity due to trauma.

- *Etiology:*

- 1) **Blunt trauma**

- Starts posterior subcapsular due rupture of the post. Capsule(thinnest part) allowing the entry of aqueous .
- Rosette or feathery shaped due to delineation of the lens fibers by the aqueous with disruption of the architecture of the cortical sutures.
- Progressive
- Vossiu's ring could be seen due to imprint of the posterior iris pigment on the anterior lens capsule.

2) Perforating trauma:

- Due to rupture of the capsule the lens ptns may escape to the AC leading:
- Severe uveitis(phacoanaphylactic or phacotoxic)
- Secondary glaucoma

3) IOFB:

- Iron: siderotic cataract +/- rust spots on the anterior capsule.
- Copper: sunflower cataract (chalcosis)

• *Management:*

According to the age. With:

1) Proper preoperative examination (meticulous ocular exam and US if cataract is dense) :
retinal detachment,vitreous he or IOFB.

2) In case of uveitis with blunt trauma :atropine and steroids are given and operation is done when the eye is quite.

3) Surgical ttt: **when the eye is quite (No I.C or glaucoma) :**

- If the patient is less than 25 years : I.A. or lensectomy
- If the patient is more than 25 years :Phaco or ECCE.

2) A regular postoperative follow up to detect late effect of trauma.

8. Causes, Types, Clinical picture of ectopialentis:

• *Etiology: (all types)*

a) *Congenital:*

Marfan's Syndrome (usually upper)

b) Acquired:

1. Traumatic especially blunt trauma.
2. Hyper-mature cataract.
3. Pseudo-exfoliation syndrome.
4. High axial myopia.
5. Buphthalmos.
6. Metabolic e.g. homocystinuria.(usually lower)

- *Types:*

- 1- Sub-laxation (partial tearing of the zonule)
- 2- Dislocation (total tearing or absence of the zonule): Anterior and Posterior

- *Clinical picture of the sublaxation:*

Symptoms:

- 1- Drop of vision: Due to myopic astigmatism.
- 2- Uni-ocular diplopia: If the edge of lens crosses the pupil, two images will be formed. One from the phakic portion and the other from the aphakic one.

Sign:

- 1- AC: Irregular depth.
- 2- Tremulous iris.
- 3- Edge of the lens may be seen from the pupil.

- **Complication:**

- 1-dislocation
- 2-secondary glaucoma
- 3-uveitis
- 4-cataract

- **Treatment:**

-if not complicated: glasses

-if severe or complicated: extraction by scoop (ICCE) and anterior vitrectomy or lensectomy

- *Clinical picture of anterior dislocation:*
the lens is seen in the AC + myopic shift
 - Complications:
 - 1- Primary glaucoma: Pupillary block glaucoma (**Glaucoma Inversus**) that worsens with miotics and relieved by mydriatics.
 - 2- Iritis.
 - 3- Corneal de-compensation and irreversible edema.
 - 4- Cataract
 - Treatment:

Immediate lens extraction with a scope (ICCE) with a limbal.
- *Clinical picture of posterior dislocation:*
 - 1- Aphakic signs. (jet black pupil, Tremulous iris and one purkinje's image).
 - 2- Drop of vision due hypermetropic shift and loss of accommodation
 - 3- Lens may be seen in posterior segment by fundus examination.
 - Complication:
 - 1-Uveitis esp. if opened capsule
 - 2-secondary glaucoma due pupillary block by vitreous
 - 3-cataract
 - Treatment if complicated

9. Give an account on: Clinical picture and correction of aphakia.

Definition:

Absence of the crystalline lens in the pupillary area. If an artificial IOP is implanted it's known as pseudo-phakia.

Etiology:

- Surgical:
 - After cataract surgery
 - Removal of a subluxated or dislocated lens

- Refractive clear lens extraction in the treatment of high axial myopia
- Posterior dislocation (DD)

Symptoms

Defective vision (esp. Near due to loss of accommodation & hypermetropic shift)

Signs:

1. JET BLACK pupil.
 2. DEEP anterior chamber.
 3. The iris is tremulous , may be with an iridectomy if ICCE.
 4. ONE corneal Purkinje-senson image.
- ❖ Purkinje-senson image are the reflections formed by the reflecting surfaces of the eye
 - ❖ Using a torch, we can clinically detect one image from the corneal surface ,one from the anterior surface of the lens , and one from the posterior surface of the lens . Those from the cornea and the anterior surface of the lens are virtual and erect and so they move with the movement of the light . The one from the posterior surface of the lens is true and inverted and so it moves against the movement of the object .
 - ❖ In aphakia there is loss of the 2 images belonging to the lens .
5. Loss of Accommodation.
 6. Optical condition after lens extraction by retinoscopy :
 - hypermetropic shift (+10 to +12 D if previously emmetropic OR if previously -25 myope will be emmetropic.
 - Astigmatism (against the rule about 2D if upper ECCE OR with the rule if temporal incision was made)
 - Anisometropia if unilateral aphakia.
 7. Limbal scar if SURGICAL OR lens seen by fundus or US if TRAUMATIC posterior dislocation.

Correction of aphakia :

1. BILATERAL:
 - IOL (1ry at time of surgery OR 2ry Anterior chamber or posterior chamber)
 - Contact Lens(rarely tolerable due to lack of accommodation and difficult dexterity in old age)
2. UNILATERAL:

- contact lens or IOL

❖ *NEVER GLASSES IN UNILATERAL CASES to avoid diplopia caused by associated Anisokonia (33%)*

10.Compare: Between the options for optical correction of unilateral aphakia

- Glasses give alarheimage , aberrations , prismatic effect and alimited field . They can not be used in the correction of unilateral aphakia to avoid diplopia caused by the difference of the size of the image on the retina between the 2 eyes (Anisokonia)
- Contact lenses are rarely tolerable due to lack of accommodation and difficult dexterity in old age.
- Primary intraocular lens implantation in the posterior chamber at the time of surgery is the conventional approach.

Secondary implantation may be resorted to , where the lens may be implanted in the anterior chamber or in the posterior chamber.'

11.Differential diagnosis of leucocoria or white pupil: (Very important):

- Retinoblastoma (M.S)
- Congenital cataract (M.C)
- Cyclitic membrane
- Coats disease (retinal telengectasia with extensive lipid exudation and exudative retinal detachment.
- Persistent hyperplastic primary vitreous
- Toxocara endophthalmitis
- Others: retinal detachment, retinal dysplasia

12.Define diplopia, mention types & causes

- Diplopia → double vision

- Types → monocular "uni "or Binocular diplopia
- Causes of monocular:
 - a. Early cortical cataract
 - b. Iridodialysis
 - c. Subluxation of lens
 - d. Anisometropia "if corrected with eye glasses"
 - e. Unilateral aphakia "if corrected with eye glasses"
- Causes of binocular diplopia:
 - a. Paralytic squint
 - b. Myasthenia gravis
 - c. Proptosis
 - d. 3rd, 4th, 6th nerve palsy

MCQ:

1) Symptoms of early senile cataract includes:

- a) Pain and headache
- b) Uniocular diplopia
- c) Transient diminution of vision
- d) Floating Opacities

Answer:(b) uniocular diplopia

2) Hypermature cataract can cause the following complications except:

- a) Phacomorphic glaucoma
- b) Phacotoxiciridocyclitis
- c) Opacification of lens
- d) Phacolytic glaucoma

Answer: (A) phacomorphic glaucoma (caused by intumescent cataract)

3) Correction of unilateral aphakia with glasses will result in :

- a) Astigmatism
- b) Glucoma
- c) Hypermetropia
- d) Anisokonia

Answer: (D) Anisokonia

4) Loss of accommodation may be found in:

- a) 3rd nerve palsy
- b) Presbyopia
- c) Tropicamide eye drops instillation
- d) All of the above

Answer: (D) All of the above

5) Best option for mannagment of anisometropia in unilateral aphakia:

- a) Glasses
- b) IOL implantation
- c) Contact lenses
- d) Radial keratotomy

Answer: (B) IOP implantation

6) After cataract is treated by:

- a) YAG laser
- b) Argon laser

- c) Excimer laser

Answer: (A) YAG laser

7) Diminution of vision in lens subluxation is caused by:

- a) Myopia
- b) Astigmatism
- c) Complications
- d) All of the above

Answer: (D) All of the above

8) The typical type of steroid induced cataract is:

- a) Nuclear sclerotic
- b) Posterior polar
- c) Posterior subcapsular
- d) Cortical

Answer: (C) Post Sub-capsular

9) Posterior dislocation of the lens is characterized by:

- a) loss of one purkinjesanson image
- b) uveitis may occur as a complication
- c) neovascular glaucoma is a complication
- d) the ocular refraction shifts towards myopia

Answer: (B) uveitis may occur as a complication

10) Vossiu's ring is found in anterior lens capsule in:

- a) complicated cataract

- b) congenital cataract
- c) traumatic cataract
- d) hypermature cataract

Answer: (C) traumatic cataract

11) Glaucoma caused by anterior lens dislocation is called:

- a) glaucomainversus
- b) pseudo-exfoliation glaucoma
- c) malignant glaucoma
- d) phacolytic glaucoma

Answer: (A) glaucoma inversus

12) Nuclear cataract changes the refraction of the eye into:

- a) Myopia
- b) Hypermetropia
- c) Astigmatism
- d) No change

ANSWER: (A)

13) Most common cause of diminution of vision after ECCE is:

- a) Cystoid macular edema
- b) Posterior capsule opacification
- c) Corneal decompensation
- d) Retinal detachment

ANSWER: (B)

14) The best ttt of Posterior capsule opacification:

- a) Surgical excision
- b) Laser opening
- c) Surgical polishing
- d) Leave alone

ANSWER: (B)

15) The type of laser used to treat Posterior capsule opacification:

- a) Yag laser
- b) Argon laser
- c) Diode laser
- d) Excimer laser

ANSWER: (A)

16) Posterior polar cataract markedly affects vision because:

- a) Its shadow lies on the macula.
- b) Close to the nodal point.
- c) It matures early.
- d) It blocks the papillary area.

ANSWER: (B)

17) All of the following are lens induced glaucoma except:

- a) Phacomorphic glaucoma.
- b) Phacoanaphylactic glaucoma.
- c) Phacolytic glaucoma.
- d) Neovascular glaucoma.

ANSWER: (D)

18) All the following are signs of lens subluxation except:

- a) Phakodonesis.
- b) Iridodonesis.
- c) Irregular anterior chamber.
- d) Intact all zonule.

ANSWER: (D)

19) The term “mature cataract” means:

- a) A nuclear cataract present more than 10 years.
- b) A posterior subcapsular cataract that reduces visual acuity to 6/60 or worse.
- c) A cortical cataract that involves the entire cortex.
- d) An anterior sub capsular cataract that causes capsular wrinkling.

ANSWER: (C)

20) Criteria of mature senile cataract:

- a) Visual acuity HM.
- b) Absent RR.
- c) Absent iris shadow.
- d) All of the above.

ANSWER: (D)

21) Tremulous iris can be seen in:

- a) Aphakia.
- b) Lens subluxation.

- c) Hypermature cataract.
- d) Posterior lens dislocation.
- e) All of the above.

ANSWER: (E)

Vitreous

GIVE ACCOUNT ON:

1. Causes of vitreous hemorrhage:

- 1- PDR
 - 2- CRVO-BRVO
 - 3- Trauma
 - 4- Vasculitis e.g. sarcoidosis.
 - 5- Blood diseases e.g. Leukemia
 - 6- Retinal tear
 - 7- Intraocular tumours.
-

Uvea

GIVE AN ACCOUT

1. anatomy of the iris

Gross anatomy:

- Forms the anterior part of the uveal tract, it is avascular diaphragm that has a central perforation called the pupil.
- It divides the anterior segment into anterior and posterior chambers.

- **Color:**

At birth, it's light blue-gray in color. The definitive color is reached at about 1 year.

- **Borders:**

- 1) Central free papillary border.
- 2) Attached ciliary border.

- **Surface:**

1) Anterior surface (iris pattern):

a. Elevations:

i. Collarette:

1. Irregular zigzag circular ring, 1.5 mm from the pupillary border.
2. It separates the anterior surface into pupillary zone & ciliary zone.

ii. Radial streaks:

Extending from the pupillary border to the ciliary border (corresponding to the radial blood vessels in the stroma).

b. Depressions:

i. Crypts:

Depressions near collarette and the ciliary border.

ii. Circular furrows:

Near the ciliary border.

2) Posterior surface:

Smooth and darkly pigmented.

Minute anatomy:

1) Stroma: Consists of CT containing:

- A. Pigmented cells (melanocytes).
- B. Radial blood vessels (radially).
- C. Nerves.
- D. Muscles:

*i. **Sphincter (constrictor) pupillae:***

- Circular fibers at the pupillary edge.
- Supplied by parasympathetic fibers of the 3rd cranial nerve.
- Contraction $\Rightarrow$ miosis.

*ii. **Dilator pupillae:***

- Radial fibers extending from the root of the iris.
- Supplied by sympathetic fibers.
- Contraction $\Rightarrow$ mydriasis.

2) Posterior epithelium:

2 layers of pigmented cells (anterior flat & posterior cubical).

Nerve supply:

1) Sensory:

Long ciliary nerves along the naso-ciliary branch of 5th cranial nerve.

2) Motor:

Short ciliary nerves:

- a. Parasympathetic: from the 3rd cranial nerve, which supplies the sphincter.
- b. Sympathetic: From the cervical chain, which supplies the dilator muscle.

Blood supply:

*1) **Circulus iridis arteriosis major:***

At the root of the iris. Formed by union of anterior ciliary arteries and 2 long posterior ciliary arteries.

*2) **Circulus iridis vasculosis minor:***

Anastomosis at the collarette.

Function:

- 1) Controls the amount of light entering the eye.
- 2) Miosis increases depth of focus.
- 3) Decreases prismatic (spherical) aberration of the lens periphery.

2. Describe etiology, signs, symptoms and treatment of acute Iridocyclitis.*Etiology:*

- **Exogenous:**

Endophthalmitis (post-surgical or open globe injury)

- **Endogenous:**

1. **Non-infective:**

- Associated with systemic diseases:
 - A) Arthritis: HLA B 27+vespondylo-arthropathies e.g. AnkylosingSpondylities.
 - B) Granulomatous diseases: e.g. sarcoidosis: middle aged black female with non-caseating granuloma affecting mainly the lungs(90%)
 - C) Behcet's syndrome: usually affects young adult males: recurrent aphthus oral and genital ulcers, recurrent uveitis with hypopyon and retinal phlebitis.
 - D) Vogt-koyanagi- Harada syndrome:Common in south east Asia, characterized by:
 - a. Recurrent Iridocyclitis.
 - b. Exudative choroiditis that leads to exudative retinal detachment.
 - c. Systemic feature (alopecia-poliosis-vitiligo-deafness).
- Associated with other ocular diseases (secondary uveitis):
 - A) Keratits
 - B) Scleritis
 - C) Ectopialentis
 - D) Following intraocular surgery or penetrating trauma
- Masquerade syndrome (fundus):

- A) IOFB
- B) IO tumour
- C) Retinal detachment

- Drugs or idiopathic

2. **Infective:** as syphilis, TB, Toxoplasmosis

Symptoms:

- 1) Ocular pain occurs due to irritation of the 5th nerve endings & spasm of the ciliary muscles.

* The iris is richly supplied with sensory nerves from the ophthalmic division of the 5th nerve. The pain is dull aching in character (neuralgic), referred to the eyebrows, and more intense at night.
- 2) Headache in the forehead and around the eye.
- 3) Lacrimation, photophobia & blepharospasm.
- 4) Redness.
- 5) Decreased visual acuity; due to:
 - a. Edema of the cornea.
 - b. Turbidity of the aqueous & vitreous
 - c. Spasm of accommodation causing myopia of 1-2 D.
 - d. Toxic maculopathy due to diffusion of PGs into the posterior pole.
 - e. Keratic precipitates on the back of the cornea and pigment deposition on the anterior lens capsule.

Signs:

1) *Circumcorneal ciliary injection:*

dilation of the anterior ciliary blood vessels and mild lid edema.

2) *VA:*

Dropped.

3) *Cornea:*

- a. Corneal edema.
- b. Keratic precipitates (KPs): inflammatory cells deposited on the endothelium by

convection currents.

- Shape $\Rightarrow$ usually triangular base down.
- Site $\Rightarrow$ Lower third (scattered in viral endothelial dusting).
- Color:
 - Fresh $\Rightarrow$ white.
 - Old $\Rightarrow$ Pigmented.
- Size:
 - Small: PMNLs.
 - Medium sized: lymphocytes,

4) *Lens:*

Lenticular iris pigment precipitates.

5) *Anterior chamber:*

- a. Aqueous flare (earliest sign) $\Rightarrow$ disruption of blood aqueous barrier with excessive protein leakage.
- b. Aqueous cells or floaters.
- c. Hypopyon: sterile pus at the bottom of AC.
- d. Hyphemia.

6) *Anterior 1/3 of vitreous:*

Flare & cells.

7) *Iris:*

Loss of the iris pattern (muddy).

8) *Ciliary body:*

Tender on lid pressing,

9) *Pupil:*

Small & sluggish (toxic myositis & straightening of iris vessels).

10) *IOP:*

Early:

Hypotony due to cyclitis (shock stage) $\Rightarrow$ shut down.

Later:

Expected to **increase** due to:

- a. Plasmoid aqueous.

- b. Trabeculitis (viral).
- c. Steroids.

11) Fundus:

- a. Toxic optic papillitis & cystoid macular edema.
- b. Exclude masquerade (secondary) cause.

Treatment:

A. Local treatment:

1) Topical cyclopentolate: as Atropine sulphate:

- A) It's the most important line.
- B) 1-2% drops in adults & 1% ointment in children. Eye drops should be avoided in children to avoid systemic toxicity. It's used 3 times daily.
- C) Mechanism of action:
 - Cycloplegic: Atropine relieves pain and headache from ciliary spasm.
 - Mydriasis: dilates the pupil and prevents posterior synechia & decreases exudation from iris vessels.

2) Corticosteroids:

- A) Prednisolone acetate 1% or dexamethasone phosphate 0.1% in the form of drops or ointment.
- B) Used frequently every 1-2 hours in the acute stage and tapered gradually.
- C) Mechanism of action:
 - Decrease the release of inflammatory mediators by stabilization of lysosomal membranes.
 - Decrease vascular permeability.
 - Decrease release of destructive leucocytic enzymes.
 - Inhibit fibroblastic activity & diminish the formation of synechiae.
 - Anti-allergic.
- D) Corticosteroids are not used in infectious cases, where the appropriate antibodies should be used instead.
- E) Side effects occur with prolonged use:
 - i. Complicated cataract: posterior subcapsular cataract (steroid-induced).

- ii. Secondary open angle glaucoma.
- iii. Reactivation of herpes simplex keratitis.

3) *Hot fomentations:*

Decrease pain & improve the circulation.

4) *Dark glasses:*

To decrease photophobia.

B. Systemic treatment:

In severe cases.

1) Steroids:

- Full dose: 1 mg/kg/d, together with an antacid.
- Indication: bilateral and severe cases.
- Contra-indications:
 - i. Infective cases (unless undercover).
 - ii. Peptic ulcer.
 - iii. Osteoporosis.
 - iv. Cardiac or renal disease (water retention).
 - v. DM.
- Side effects:
 - i. Hypokalemia.
 - ii. Hyperglycemia.
 - iii. Salts & water retention.
 - iv. Acute gastric erosions.
 - v. Psychosis.

2) NSAIDs:

- Action: inhibit PG release.
- Indication:
 - i. As an adjuvant to steroid therapy.
 - ii. Contraindication to steroid therapy.

3) Immunosuppressive drugs:

In steroid resistance cases: Cyclosporine in Behcet.

4) Analgesics.

5) Anti-infective agents:

If infective. e.g. Syphilis, toxoplasmosis endophthalmitis.

C. Treatment of the cause:

D. Treatment of complications:

- 1) Cataract $\Rightarrow$ after 6 months of a uveitis-free period.
- 2) Secondary open angle glaucoma $\Rightarrow$ add to the ttt BB and CAI, but never Pilocarpine.
(Prostaglandin analogues are contraindicated in cases of uveitis).
- 3) Closed angle glaucoma (PAS): subscleraltrabeculectomy after 6 months of uveitis-free period.
- 4) Cyclitic membrane $\Rightarrow$ vitrectomy and membranectomy if good retinal function.
- 5) Ring synechia $\Rightarrow$ peripheral iridotomy.
- 6) Atrophic bulbi $\Rightarrow$ enucleation and artificial eye.

3. Discuss complications of Iridocyclitis.

1. Organization of exudates:

A. Between the iris & the lens (*posterior synechiae*):

- a. Partial: festooned pupil on dilation.
- b. Ring or annular: seclusio-pupillae.
- c. Total: adhesion plastering the whole posterior surface of the iris & the lens.

* Both seclusio and total synechiae might cause secondary closed angle pupillary block glaucoma. The only difference would be the iris configuration (by the presence of iris bombe with seclusio).

B. At the pupil:

Pupillary membrane (occlusio-pupillae).

C. At the angle:

Peripheral anterior synechiae.

D. Behind the lens (*retrolental space*):

Cyclitic membrane.

- a. Appears as a yellow-white reflex at the pupil (DD of leukocoria).
- b. May pull on the ciliary body leading to ciliary body detachment and hypotony
leading to atrophie,
- c. May pull on the vitreous leading to tractional retinal detachment.

2. *Secondary glaucoma:*

A. *Early: (2^{ry} open angle)*

- a. Plasmoidaqueous.
- b. Trabeculitis esp. viral.
- c. Steroid intake if responder.

B. *Late: (2^{ry} closed angle)*

- a. PAS.
- b. Seclasio-pupillae.
- c. Occlusio-pupillae.
- d. Neo vessels (retina).

3. *Secondary (complicated) cataract:*

The most common complication. Starts as posterior subcapsular with a polychromatic luster & progressive. it could also be steroid induced in chronic cases.

4. *Posterior segment complications:*

- a. Spread of toxins: cystoid macular edema.
- b. Spread of inflammation: chorio-retinitis.
- c. Retinal detachment:
 - i. Exudative from choroiditis.
 - ii. Tractional from cyclitic membrane.

5. *In long standing chronic uveitis:*

- a. Band keratopathy: calcium deposition in the Bowman's membrane.
- b. Iris:
 - i. Atrophy.
 - ii. Ectropion uvea.
 - iii. Rubeosis with secondary neovascular glaucoma.

- c. Absolute glaucoma.
- d. Atrophiabulbi (soft shrunken eye due to decreased aqueous production).

4. Mention 4 systemic diseases associated with Iridocyclitis.

1. Arthritis:

e.g. Ankylosing spondylitis, Reiter's syndrome, collagen vascular diseases.

2. Granulomatous diseases:

e.g. Sarcoidosis: usually affects middle aged black females with non-caseating granuloma affecting mainly the lungs (90%), skin, CNS & granulomatous pan-uveitis.

3. Behcet's syndrome:

Usually affects young adult males from the Eastern-Mediterranean region, characterized by:

- a. Recurrent aphthous oral & genital ulcers.
- b. Recurrent uveitis with hypopyon.
- c. Retinal phlebitis (may lead to CRVO).

4. Vogt-Koyanagi-Harada syndrome:

Common in south east Asia, characterized by:

- a. Recurrent iridocyclitis.
- b. Exudative choroiditis that leads to exudative retinal detachment.
- c. Systemic feature (alopecia-poliosis-vitiligo-deafness).

5. Discuss Endophthalmitis:

Definition:

Acute suppurative inflammation confined to the intra-ocular tissues (middle coat/inner coat and adjacent cavities). the outer coat and tenon's capsule are free.

Etiology:

1) infective:

A) Exogenous:

- Post-operative: acute (most common) or delayed common after glaucoma surgery.

- Post trauma(penetrating): if delayed antibiotics.
- Perforation of corneal ulcer.

B) Endogenous(metastatic): in immunocompromised patients

2) Non-infective:

Post-operative sterile endophthalmitis e.g. chemicals as glove powder.

Causative agent(in infective):

- 1) Acute onset(bacterial): staph epidermis, staph aureus,...
- 2) Delayed onset:
 - Propionibacterium acnes: responsible for delayed onset endophthalmitis esp. after cataract surgery(presents with a capsule plaque)
 - Fungal: candida, Aspergillus.

Clinical picture:

a) Symptoms:

As uveitis but more severe and the vision is dropped up to no PL.

b) Signs:

- 1) As uveitis but more severe.
- 2) Hypopyons (infected not sterile)
- 3) Vitreous abscess (yellow reflex)

c) Complication:

- 1) Spread of infection:
 - Panophthalmitis
 - Orbital cellulitis
 - Brain spread (CST-meningitis)
- 2) Healing that ends in phthisis bulbi.

Management:

- Hospitalization
- Investigation:

- Anterior chamber and vitreous sampling for culture and sensitivity & gram + Giemsa stain.
- Ultrasonography
 - 1) to confirm diagnosis (vitreous abscess)
 - 2) Exclude RD.
 - 3) Exclude foreign body in traumatic cases.

Treatment:

If the vision is PL or better:

- *In bacterial:*
 - 1) Vitrectomy and intravitreal antibiotics: Vancomycin for G+ve and Ceftazidime for G-ve.
 - 2) Topical: Vancomycin and ceftazidime
 - 3) Subconjunctival: vancomycin and ceftazidime.
 - 4) systemic: Ciprofloxacin

N.B.: Steroids may be added after at least 24 hours of intensive antibiotic therapy.

- *In Fungal:*
Vitrectomy and intra-vitreous amphotericin B with systemic and topical antifungal (monitor kidney functions)

If the vision is No PL:

Evisceration better than enucleation.

D.D.: panophthalmitis-red eye syndrome.

6. Discuss Panophthalmitis:

Definition:

Acute suppurative inflammation of all coats of the eye including the outer coat & Tenon's capsule.

Etiology: causative agent: as endo.

Clinical picture:

a) Symptoms:

Asendophthalmitis and the vision dropped up to no PL + fever (pyrexia), AHM, rapid pulse.

b) Signs:

As endophthalmitis +

1. Slight proptosis due to tenonitis.
2. Total ophthalmoplegia.
3. Pus in the cornea (corneal abscess) that might end in self-evisceration (perforation) when it bursts.

c) Complication: as endophthalmitis*Treatment:*

Evisceration and antibiotics (extensive topical & systemic).

* Enucleation is contraindicated as it may allow extension of infection to the brain (meningitis/CST).

Differential diagnosis:

- 1) Acute painful proptosis:
 - a. Panophthalmitis.
 - b. Orbital cellulitis.
 - c. Cavernous sinus thrombosis.
- 2) Endophthalmitis.
- 3) Red eye syndrome.

7. Enumerate The ocular side effects of topical and systemic corticosteroids therapy:

Side effects of topical steroids:

- 1) Glaucoma
- 2) Posterior subcapsular cataract
- 3) Infections: reactivation of herps
- 4) Delayed wound healing

Prolonged systemic use:

- 1) Peptic ulcer
 - 2) Diabetes
 - 3) Hypertension
 - 4) Cataract
 - 5) Osteoporosis
 - 6) Muscle wasting
 - 7) Reactivation of TB
 - 8) Cushinoid
 - 9) Acute adrenal insufficiency if suddenly stopped
-

MCQS:**1. Treatment of Iridocyclitis can include all of the following, except:**

- a) Systemic steroids.
- b) Topical steroids.
- c) Vasodilators.
- d) Cycloplegics.

Answer: (C) Vasodilators.

2. The following investigations are useful in a case of uveitis, except:

- a) Chest X-ray.
- b) Rheumatologic assessment.
- c) Echocardiography.
- d) Tuberculin test.

Answer: (C) Echocardiography.

3. The role of surgery in uveitis includes all of the following, except:

- a) Peripheral iridotomy in the presence of pupillary block.
- b) Argon laser photocoagulation in the presence of exudative retinal detachment.
- c) Cataract extraction in the presence of complicated cataract.
- d) External fistulizing surgery in the presence of chronic glaucoma.

Answer: (D) External fistulizing surgery in the presence of chronic glaucoma.

4. Treatment of acute iridocyclitis should not include:

- a) Cortisone drops & ointment.
- b) Pilocarpine 2% eye drops.
- c) Atropine 1% eye drops.
- d) Systemic steroids.
- e) Treatment of the cause.

Answer: (B) Pilocarpine 2% eye drops.

5. Complications of iridocyclitis include:

- a) Ghost cell glaucoma.
- b) Dislocated lens.
- c) Iris bombe.
- d) Anterior polar cataract.
- e) Monocular diplopia.

Answer: (B) Dislocated lens.

6. In endophthalmitis, the red reflex becomes:

- a) Reddish.
- b) Whitish.
- c) Yellowish.
- d) Black.

Answer: (C) Yellowish.

7. Enucleation can be done in panophthalmitis:

- a) True.
- b) False.

Answer: (B) False.

8. Keratic precipitates seen in iridocyclitis are inflammatory cells deposited in:

- a) Corneal epithelium.
- b) Corneal endothelium.
- c) Lens endothelium.
- d) Retinal pigment epithelium.

Answer: (B) Corneal endothelium.

9. Secondary glaucoma due to acute iridocyclitis should not be treated by:

- a) Atropine 1%.
- b) Topical & systemic corticosteroids.
- c) Pilocarpine 2%.
- d) Diamox.

Answer: (C)Pilocarpine 2%.

10. Enophthalmos may be due to:

- a) Trauma.
- b) Cachexia.
- c) Post radiotherapy.
- d) Secondaries of breast scirrhous carcinoma
- e) All of the above.

Answer: (E)

11. The pupil in acute anterior uveitis is :

- a) Constricted
- b) Dilated
- c) Festooned
- d) Vertically oval

Answer: (A)

Glaucoma

GIVE AN ACCOUNT

1. Define glaucoma and how would you classify it:*Definition:*

It's a progressive optic neuropathy having a rise of IOP as a major risk factor.

*Classification:***According to the age:**

- A) Congenital
- B) Acquired

According to the presentation (Etiology):

- A) Primary

B) Secondary

N.B: all primary glaucomas are bilateral and familial.

According to the angle:

A) Open

B) Closed (by iris): Peripheral anterior synechiae(PAS).....Partial or total

N.B: this classification is practically very useful since ttt of

- Open angle glaucomas is initially medical.
- Closed angle glaucomas is essentially surgical.

2. Discuss Buphthalmos (primary congenital glaucoma):

Definition:

Rise of IOP before 2-3 years (due to an elastic outer coat) with enlargement of the globe (buffalo or ox lite eye), due to a congenital angle anomaly.

Etiology:

Angle anomaly

- 1- Barkan's membrane (mesoderm obstructing the angle)
- 2- Anterior insertion of the ciliary muscle in the TM
- 3- Dys-genesis of the schlemm's canal (a consequence rather than a cause)

Now believed to be due to an isolated trabecular dys-genesis characterized by narrow spaces of Fontana with flat anterior insertion of iris in the TM, possibly due to delay of AC angle development

Clinical picture:

Incidence:

1\10000 (more common when parents are relatives)

Demography:

- Age: before 3 years, neonatal (40%) or infantile
- Sex: boys (2:1)
- Laterality: bilateral (75-80%) usually asymmetrical
- Family history: positive (AR)

Symptoms:

As given by mother.

- Early: triad of blepharospasm, lacrimation and photopia
- Late: Blue sclera, large globe and poor vision (Cloudy cornea)

Signs:**I. Signs of a big eye (buphthalmous):**

- 1- **External appearance** → pseudo-proptosis or pseudo-exophthalmos
- 2- **Cornea:**
 - a) Diameter: horizontal corneal diameter (HCD): increased up to 13-14 mm (N: 10 at birth reaching adult size 11.75mm at 1 year)
 - b) Haab's Stria: horizontal Stria due to healed curvilinear breaks in descemet's membrane
 - c) Transparency: Haze due to edema from aqueous influx
 - d) Curvature: decreased (more flat and thin)
- 3- **Sclera** → is thin and blue (stretching with visibility of underling uvea)
- 4- **Anterior chamber** → Deep
- 5- **Iris** → Tremulous (iridodonesis)
- 6- **Pupil** → dilated and sluggish
- 7- **Lens** → flattened and displaced backwards
- 8- **Zonule** → stretched may lead to ectopia lentis
- 9- **Refraction and axial length** → axial myopia + \- astigmatism:
due to globe enlargement and corneal irregularity, but less than expected due to flattening of the cornea and flattening with posterior displacement of the lens
(normally every 1mm: 3D)
- 10- Appearance of a cause in primary cases, i.e. exclude secondary

II. SIGNS OF GLAUCOMA:

- 1- **IOP** → high but less than expected because of the elastic outer coat
- 2- **Gonioscopy** → angle anomaly
- 3- **Fundus**:

- a) Cupping either due to loss of nerve fibers and/or widening of the scleral canal which could be **reversible**
- b) Exclude retinoblastoma (*Any Buphthalmos is retinoblastoma UPO*)

Complications:

- 1- Glaucomatous optic atrophy (absolute glaucoma)
- 2- Lens:
 - a) Ectopia lentis (sub-laxation or dislocation)
 - b) Secondary cataract
- 3- Corneal scarring
- 4- Amblyopia (squint in unilateral cases or nystagmus in bilateral cases)

Treatment:

ESSENTIALLY SURGICAL

As medical ttt is not effective and safety unknown, used only as temporary measure before surgery

- a. Clear cornea = Goniectomy (Ab-interno):
operation of choice when feasible, and could be repeated
- b. Hazy cornea = Sub-scleral trabeculotomy (Ab-externo):
can be also done if failed Goniectomy
- c. Advanced cases (IOP>33, HCD>13 Or failed trabeculotomy):
 - ❖ Sub-scleral trabeculectomy: (External filtering surgery = filtering surgery) with or without anti-metabolites
 - A Peripheral iridectomy is also done to avoid obstruction of the sclerostomy and pupillary block glaucoma
 - Anti-metabolites could be used to decrease late bleb failure induced by fibrosis that might hinder aqueous filtration
 - ❖ Glaucoma drainage device (valve as Ahmed's valve or shunt or tube or stone) surgery

N.B:

- *The mother should be warned that her child should avoid trauma, e.g. contact sports, as his eyes are fragile*
- *The associated amblyopia should be treated*

N.B: Striae of the cornea:

<i>Haab's Striae</i>	<i>Vogt's Striae</i>
- Horizontal - Descemet's membrane - Buphthalmos	- Vertical - Stroma - Kerato-conus

3. Discuss the acute stage of Primary closed angle glaucoma: (V.V.IMP)

Pathogenesis:

- sudden total closure of the angle by peripheral Iris
- Iris sphincter ischemia from the very high pressure prevents the physiological miosis

Clinical Picture:

Symptoms:

- 1) Pain:
 - a) rapid bursting (due to stretch of outer coat)
 - b) with reflex BLP and sometimes reflex central vomiting +/- acute abdomen (vagal stimulation)
- 2) Defective vision due to:
 - a) corneal edema
 - b) optic nerve ischemia
- 3) Redness +/- rainbow colored haloes around lights

Signs:

Swollen lid +

- 1) VA: severe drop up to counting fingers (CF) hand motion (HM) or even PL
- 2) circum corneal ciliary congestion
- 3) cornea: epithelial edema +/- bullae
- 4) anterior chamber: shallow (up to irido-corneal contact = lost AC)
- 5) Iris: bombe and congested
- 6) Pupil:
 - semi-dilated
 - fixed irreactive
 - vertically oval (segmental innervation of iris muscles)
 - green-blue
- 7) IOP: stony hard (digitally)

Treatment:

Surgical after medical treatment

It's an ocular emergency that requires immediate hospitalization and lowering of the IOP (risk of optic nerve ischemia and central retinal artery occlusion if left untreated)

I. Medical Preparation:

- Aim: Lower IOP to avoid expulsive hge and decrease intraocular inflammation

1) Hyperosmotic agents:

e.g. mannitol 20% 1gm/kg 60 drop/min over 30 minutes IV drip

mechanism: withdraws water dehydrating the vitreous

N.B. avoid if cardiac, hypertensive, renal, and prostatic enlargement due to volume overload

2) Acetazolamide:

carbonic anhydrase inhibitor = Diamox

500 mg IV then 250 mg oral qid (cidamex) +/- beta blocker eye drops (timolol)

mechanism: decrease aqueous secretion

N.B. avoid Diamox in sulpha allergy

3) Miotics:

pilocarpine 1-4% eyedrops every 5 min until constriction then qid

Mechanism: contraction of the sphincter with mechanical pulling of the iris from the angle (main action in PACG)

N.B. miotics should be only given after the IOP is lowered by mannitol

4) Topical anti-inflammatory steroid eye drops (qid):

mechanism: to decrease inflammation and synechia (PAS) formation

+/- systemic analgesics / anti-emetics

II. Gonioscopy:

To evaluate the angle for peripheral anterior synechiae (PAS) after lowering the IOP and clearing of the cornea and pain to decide the surgical treatment

III. Surgical treatment:

- No or mild PAS and within 24 hrs. = upper peripheral Nd-YAG iridotomy
- If extensive PAS occluding the angle = sub-scleral trabeculectomy (SST)

N.B. if surgery is not done the patient may turn into chronic ACG

IV. Prophylactic TTT for the other eye:

Peripheral upper laser iridotomy

Fate Of The Attack:

- if untreated: the vision might be lost due to optic N. atrophy (absolute stage) or CRAO
- if treated medically only: without surgical opening of the angle, it may turn into the chronic stage with progressive optic N. damage
- post congestive stage (Posner's triad):
 - 1) patches of iris atrophy
 - 2) pigment dispersion
 - 3) glaucomoflecken: anterior sub-capsular cataract due to necrosis of the anterior sub-capsular epithelium

4. Discuss Primary closed angle glaucoma:

Definition:

Primary glaucoma characterized by closure of the angle (aqueous outflow) by the peripheral iris (partial or total)

Incidence: → 1/1000

Demography:

Age:

Above 40 years (usually above 60 years, because the lens at 40 years and above is relatively large to this small eye pushing the iris forwards)

Sex → females (4:1)

Laterality → Bilateral asymmetrical

Family history → +ve

Race → Asians because their eyes are small

Etiology:

Exaggeration of the physiological iris bombe that occludes an anatomical predisposed angle when opposed to physiological precipitating factors leading to pupillary block of aqueous flow

★ Predisposing factors (anatomical):

- Age & race.

- **High axial hypermetropia** characterized by shallow AC & narrow angle.

★ Precipitating factors (physiological):

1- Pupil dilatation:

a) Darkness.

b) Drugs (mydriatics): e.g. - fundus examination:

– sympathomimetic (e.g: adrenaline) – parasympatholytics (e.g: atropine)

2- Ciliary body congestion e.g: prone position or emotional excitation.

Clinical picture:

1- Intermittent = prodromal stage

2- Acute phase (emergency).

3- Chronic phase.

4- Absolute phase.

1) Intermittent = prodromal stage

- **Rapid partial** closure of the angle by the iris followed by re-opening by physiological miosis (such after exposure to light & sleeping).

Symptoms:

- During the attack: 1- headache 2- halos 3- blurred or foggy vision.
- In between the attacks: 1- history of headache, halos & blurred vision.
2 -Misdiagnosed as migraine.

Signs:

- During the attack:
 - 1- IOP: high, but less than 40-50 mmHg.
 - 2- Cornea: edema.
 - 3- No redness.
- In between the attacks:
 - 1- Shallow AC.
 - 2- Narrow angle in both eyes.

N.B: provocative test in the past.

Treatment:

Surgical after medical preparation

Bilateral upper peripheral LASER iridotomy or surgical iridotomy. Pilocarpine is given to both eyes as a preparation for iridotomy.

Aim: to bypass the block at the pupil allowing aqueous to flow from the PC to AC.

- Upper (12 o'clock); to be covered by the upper lid to avoid diplopia & cosmetically.
- Peripheral to avoid lens injury.
- Bilateral, because the outer eye is said to in latent phase (50% develop acute attack within 5 years if unilateral).

2) Acute phase:

See before.

3) *Chronic phase:*

Etiology:

- As consequence of medical ttt of acute attack.
- Repeated attacks of partial closure leads to accumulation a ring or "creeping PAS".

Clinical picture:

- As POAG (asymptomatic, high IOP, cupping & field changes).

Treatment:

SST-ectomy.

D.D: POAG.

	<i>POAG</i>	<i>Chronic PACG</i>
<i>1-History - sex - age</i>		
<i>2- Gonioscopy</i>	Open	Closed: PAS Other eye: narrow
<i>3- Slit lamp</i>	Normal AC or deep if myope	Shallow AC (hyperope) +/-positive congestive sign
<i>4- ttt</i>	-Initially medical. -surgical if failed.	Initially surgical (SST).

4) *Absolute phase:*

Signs:

- 1) VA: no PL (no perception of light).
- 2) Cornea: degenerative pannus & edema.
- 3) Iris: atrophic, ectropion uvea & rubeosis iridis.
- 4) Angle: +/- PAS.
- 5) Lens: cataract.
- 6) Sclera: staphyloma.
- 7) Optic nerve: glaucomatous atrophy.
- 8) IOP: high then low in the atrophic stage (atrophia bulbi).

Treatment:

If blind & painful:

- 1- Cyclodestructive: destruction of ciliary body by:
 - LASER photocoagulation
 - Diathermy
 - Cryo
- 2- Retrobulbar injection of 70% alcohol (block ciliary ganglion).

If atrophied bulbi or blind & staphylomatous (ugly):

Evisceration or enucleation & artificial eye implant.

D.D. of the acute stage of PACG:

- 1) Red eye syndrome.
- 2) Plateau iris syndrome.
- 3) Secondary ACG:
 - a. Inflammatory (uveitis).
 - b. Neovascular: rubeosis iridis.
 - c. Lens-induced:
 - Phacolytic: hypermature cataract + deep AC + fluffy white particles in the AC.
 - Phacomorphic: immature cataract + glistening of the capsule + water vacuoles in the lens.

N.B. In secondary acute congestive glaucoma, the other eye is usually normal i.e. No shallow AC and there is evidence of the cause.

5. Discuss the primary open angle glaucoma(chronic simple or compensated):

Definition:

Primary glaucoma characterized by : adult onset, open angle, high IOP, characteristic fundus and field changes with no secondary causes.

Pathogenesis:

Genetically determined age related trabecular sclerosis that leads to decrease in aqueous outflow facility.

Incidence:

1-2% the most common primary glaucoma.

Demography:

Age: old age

Sex: equal

Laterality: bilateral asymmetrical

Family history: +ve

Race: black race (most common and more difficult to treat)

Risk factor:

- Demographic factors.
- Ocular hypertension.
- **High axial myopia**
- Steroids (in responders).
- Systemic disorder as DM and vasospastic disorder (migrane).

Clinical picture:

Symptoms

- Many cases are asymptomatic and accidentally discovered.
That's why routine checking of IOP (particularly after age of 40) is the only way to discover the disease before it steals vision.
- Decreased amplitude of accommodation (pathological presbyopia): frequent changing of reading glasses.
- Some patients may complain of defective dark adaptation described by the patient as night blindness.
- Defect in visual field is very late due the fact that initially the field defect are relatively compensated by the other eye.

Signs

There are 3 cardinal signs in open angle glaucoma:

1- ELEVATION OF IOP:

It's usually elevated except in cases of normal-tension glaucoma.
Any rise of IOP above 22mmHg is suspicious.

2- CUPPING OF THE OPTIC DISC "seen in fundus examination":

The optic nerve head is the most important structure to be examined in glaucoma.
The optic nerve head is the channel through which the nerve fibers leave the globe to the brain.
It normally appears as pale pink slightly oval disc with a whitish central area known as the physiologic cup.
The normal horizontal cup-disc (C/D) ratio is usually 0.3 and the cup is usually circular and has sloping walls.

- ***Early changes of the optic disc in glaucoma:***
 - a) Large cup disc ration > 0.4
 - b) Asymmetry of C/D ratio between the 2 eyes
 - c) Vertical elongation of the optic cup
 - d) Notching of the rim of the cup
 - e) Splinter hemorrhage on the disc
 - f) Increased visibility of the pores of the lamina cribrosa
 - g) Nerve fiber layer defects with red free filter
- ***Late changes of the optic disc in glaucoma:***
 - a) Large cup with C/D ratio > 0.7
 - b) Deep cup with undermined edges
 - c) Nasal shift of the vessels

3- FIELD CHANGES: (draw the defects)

Visual field changes in glaucoma are determined by the nature of distribution of the ganglion cell axons as they converge towards the optic nerve to leave the eye.

- The most crowded fibers at the optic nerve are the temporal fibers above and below the macula (the arcuate fibers) and this explains why the earliest glaucomatous field defects occur in this zone
- The least crowded are the macular fibers and this explains the relative sparing of visual acuity until the late stages of the disease
- No fibers from the upper half of the retina enter through the lower half of the disc and vice versa and this explains why glaucomatous field defects respect the horizontal meridian
 - ***Early changes in the central field (30 degrees):***
 1. Paracentral scotoma: the earliest defect, isolated scotoma bet. (10-20) degrees of fixation, it usually starts upper nasal.
 2. Siedle scotoma: due to elongation of the paracentral scotoma along the distribution of the arcuate fibers to connect with the blind spot.
 3. Arcuate(Bjerrum scotoma): arches around the point of fixation not exceeding the horizontal raphe.
 4. Double arcuate scotoma: lower and upper arcuate scotomas fuses.

5. Ronne nasal step.

- ***Advanced late cases:***

-concentric contraction of the peripheral field.....eventually tubular field and an accompanying temporal island.

- ***End stage:*** total blindness (Absolute glaucoma).

6. Discuss the lines of TTT of POAG:

I. Medical:

First choice anti-glaucoma medications:

1- **Beta blockers:**

The prototype of this group is timolol maleate "non-selective beta blocker"

Mechanism → decrease aqueous secretion.

Dose → twice daily.

Side effects: (so contraindicated) →

Allergy-Tachyphylaxis-**Bronchospasm-Bradycardia**-Blunting of the sympathetic response to hypoglycemia in diabetics-insomnia-nightmares

2- **Prostaglandins analogues:**

The prototype of this group is latanoprost (Xalatan)

Mechanism → increases the uveoscleral outflow.

Dose → once daily at night.

Side effects →

Hyperpigmentation of the iris and preorbital skin-Lengthening and pigmentation of eyelashes-Mild iritis-cystoid macular edema in aphakia and pseudophakia.

Second choice medications:

They're added to the first-choice medications if IOP is not well controlled, the patient does not tolerate the first choice group or used for a limited period of time as postoperative or in secondary glaucoma

1- **Alpha agonists:**

The prototype is bromonidine 0.2% (Alphagan)

Mechanism → Decrease aqueous secretion and increase uveoscleral outflow

Dose → twice daily.

Side effects → Local allergy-dry mouth-fatigue_apnea in children.

2- **Miotics:**

Especially pilocarpine nitrate 1-4%

Mechanism:

- contraction of ciliary muscles will pull on the scleral spur widening the pores of the trabeculum(main action) and it compresses the blood vessels decreasing aqueous secretion.
- Contraction of the sphincter will pull the iris from the angle and will stretch the iris increasing the absorption of aqueous.

Dose → 4 times daily.

Side effects:

- Due to miosis: decrease night vision and field of vision
- Due to ciliary contraction: headache and myopic shift

N.B: never use miotics in secondary glaucoma.

3- **Topical carbonic anhydrase inhibitors:**

Dorzolamide and Brinzolamide

Mechanism → decrease the active secretion of aqueous.

Dose → 2-3 times daily.

Side effects → local allergy.

4- **Systemic carbonic anhydrase inhibitors:**

The most common drugs are acetazolamide (Diamox) and dichlorpheniramide (oratrol)

Side effects: **Parasthesia**-GIT upset- CNS depression- renal stones- blood dyscrasias.

II. Neuroprotective drugs:

Newer drugs that are under trial include the glutamate inhibitors that act as neuroprotectors decreasing ganglion apoptosis.

III. Surgical:

As SST+/- antimetabolites, Valve or Tube surgery.

Indication: Failed medical and LASER or unavailability of LASER.

- It is used primary (not preceded by medical: in very young patients with advanced disease or with patient that can't afford the cost of medication.

7. Discuss lens induced glaucoma:

I. CATARCT

A. Immature → Intumescent:

-Phaco-morphic due to absorption of aqueous by the lens fibers → pupillary block glaucoma.

Characterized by:

- 1- Rapid painful drop of vision + red eye.
- 2- Shallow AC –pushed iris.
- 3- Glistening stretched capsule.
- 4- Water vacuoles.
- 5- High IOP: 2ry PBG phacomorphic glaucoma.

B. Hyper mature:

- 1) Morgagnia : due to liquefaction of cortical material → dark brown nucleus sunken in amilky liquefied cortex → pupillary block glaucoma.

Characterized by:

- 1- Rapid drop of vision up to HM.
- 2- Color :brown nucleus sunken in a liquefied white cortex .
- 3- Iris shadow: absence.
- 4- Red reflex :absence .
- 5- Lens : swollen.
- 6- Iris : pushed.
- 7- AC : shallow.
- 8- Capsule : Calcium & cholesterol.

- 2) Phaco-lytic: FB reaction → engulfed by macrophage → block angle → 2ry OAG.

Characterized by:

- 1- Rapid drop of vision up to HM.
- 2- color : white.
- 3- Red reflex : absence.
- 4- lens : +/- shrunken.
- 5- AC : Deep lens Ptns.
- 6- Capsule : Calcium – cholesterol wrinkled .
- 7- IOP : high .

- 3) Ectopia lentis:

- subluxation → uveitis.

- Anterior dislocation → pupillary block glaucoma by lens (**glaucoma inversus**).
- posterior dislocation → pupillary block glaucoma by vitreous.

II. Capsule:

- pseudo-exfoliation.
- dandruff like material → block angle and deposits on capsule → 2ry Glaucoma.

ttt as OAG:

- Medical.
- Surgical.
- LASER.
- Associated with weak zonule → Risk during cataract surgery.

8. Mention causes & mechanisms of Glaucoma following blunt trauma of the eye.

I. *Open angle glaucoma following trauma may be due to:*

1. Traumatic iridocyclitis
2. Hyphema:
 - the blood in the AC may obstruct the pores of the trabecular meshwork and decrease the aqueous outflow from the eye (RBCs clogging the angle)
3. Ghost cell Glaucoma:
 - It occurs in long standing cases of vitreous hge where the red blood cells breakdown and their rigid walls (ghost cells) diffuse into the aqueous and obstruct the trabecular walls.
4. Angle recession Glaucoma:
 - Glaucoma following trauma secondary to tear of the ciliary body and recession of the angle structures backwards that may obstruct the aqueous outflow channels. these findings are evident by gonioscopy.

II. *Secondary closed angle Glaucoma following trauma may occur due to*

- 1-Traumatic iridocyclitis: Leading to ring synechia and occlusion pupillae.
- 2 - Organization of the blood at the angle (PAS).
- 3 -Organization of the blood at the pupil (pupillary block) .
- 4-Anterior lens dislocation: into the AC.

9. Causes of secondary open angle glaucoma.

Common features:

- 1) The angle is wide open on gonioscopy.
- 2) The AC is of normal depth.
- 3) The cause of glaucoma can be identified by examination.

Classification:

- I. **Lens induced Glaucoma** (see before)
- II. **Glaucoma secondary to intra ocular inflammation:**

1) glaucomata-cyclitic crisis:

attack of unilateral rise of IOP with a normally open angle headache & blurring of vision are common due to corneal edema.

the cause is mostly related to disturbance of PGs metabolism of the eye.

2) Uveitis:

-the causes of glaucoma is the plasmoid aqueous as well as swelling of the trabecular pores. the uveitis is managed as usual in addition to beta blockers, local & systemic carbonic anhydrase inhibitors to lower the IOP.

3) Herpes simplex & Zoster:

the causes of glaucoma are inflammation of trabecular cells.

N.B: intraocular inflammation may be complicated by secondary angle closure glaucoma due to the occurrence of peripheral anterior synechia.

- III. **Traumatic glaucoma:** (see before)

- IV. **Glaucoma secondary to intraocular tumors:**

- 1) space occupying lesion
- 2) Direct angle invasion in Iris +CB melanoma
- 3) Tumor growth >> pushing iris, lens diaphragm forwards >> secondary C.A.G
- 4) Tumor necrosis >> toxic uveitis
- 5) NV glaucoma of consumption of bl. Supply >> retinal ischemia.
- 6) ghost cell glaucoma due to vit.hge>> degenerated RBCs
- 7) Blockage of the angle by cells seedling (may lead to malignant pseudo-hypopyon) or pigment shedding in melanoma (melanomalytic glaucoma).
- 8) closure of vortical vein in the oblique sclera canals in equatorial tumors.

V. **Corticosteroid induced glaucoma:**

secondary open angle glaucoma may follow the prolonged use of certain drugs, the most common is corticosteroids due to deposition of mucopolysaccharide like material in the angle. Treatment is by stopping the steroid therapy or if steroid is absolutely necessary. topical beta blockers should be prescribed to lower the IOP.

VI. **Miscellaneous causes:**

1) Pseudo- exfoliative glaucoma :

- secondary open angle glaucoma due to excessive production of basement membrane –like material by the ocular epithelium resulting in deposition of white dandruff- like material on the surface of the lens, pupil, and trabecular meshwork.
- Blockage of the trabecular meshwork by this material obstructs the aqueous outflow from the eye.
- In these cases the zonule are usually weak:
 - Risk during cataract surgery.
 - Liable for ectopic lentis.
- The ttt is the same as POAG.

2) Pigmentary glaucoma:

due to liberation of iris pigment into the aqueous humor, with subsequent deposition in the angle and the back of the cornea (krukenberg spindle).

- It affects middle –aged males with moderate myopia.
- The pigment dispersion results from excessive rubbing of the peripheral iris against the lens zonulea.
- Transillumination defects of the iris can be seen in the patients.

10. **Causes of secondary closed angle glaucoma:**

Common feature:

- 1) Gonioscopy shows closure of the angle
- 2) The AC is shallow
- 3) The cause of glaucoma can be identified by examination.

Classification:

- 1) **Lens induced glaucomas:** see above.

2) Causes in the iris:

- the cause of glaucoma is ring synechia or total posterior synechia. Filtration surgery is indicated.
- Iris tumors or cysts.
- Pushing the iris forwards by a rapidly growing posterior segment mass or intra-ocular hge.

3) Neovascular glaucoma:

- Due to abnormal blood vessels growing from the iris surface to the angle of the AC, obstructing the TM and impairing aqueous outflow.
- CRVO is the most common cause
- **It passes into three stages:**
 - a. Stage of rubeosis iridis: the abnormal vessels are limited to the surface of the iris. It usually starts at the pupillary border of the iris
 - b. Stage of open angle glaucoma: the abnormal vessels encroach on the angle, with subsequent leakage of ptn that lead to iridocyclitis and subsequent open angle glaucoma.
 - c. Stage of angle closure glaucoma: the end stage where anterior synechia closes the angle.
 - In this type of glaucoma miotics are contraindicated to avoid ocular congestion and inflammation.
 - If discovered early, retinal pan LASER photocoagulation using argon or diode LASER controls the rise of pressure and rubeosis regresses.
 - In neglected cases, cyclodestructive procedures to diminish aqueous formation are indicated. This may be done using trans-scleral diode cyclodestruction.

11. Discuss the D.D. of Hypotony:

1. Retinal detachment
2. Disturbed outer coat
 - Rupture globe
 - Perforated corneal ulcer

3. Dysfunction of the ciliary body:

- Acute uveitis (ciliary shut down)
- Ciliary body detachment (due to cyclitic membrane)
- Ciliary body atrophy: Absolute glaucoma / Chronic Uveitis / Cyclodestructive operation e.g. cryotherapy.

4. Severe dehydration: diabetic coma.

Signs:

- Drop of vision
- Soft tension
- Descemet's fold
- Hypotonous maculopathy (choroidal folds)
- Serous choroidal detachment
- Of the cause

12. Define scotoma, mention its types and causes:

Definition:

It's an area with decreased retinal sensitivity

- ❖ Absolute: blind area
- ❖ Relative: decreased sensitivity to different intensities of light or certain colours
- ❖ Negative: not seen by the patient
- ❖ Positive: seen by the patient

Causes:

- Glaucoma
- Retinitis pigmentosa
- Blockage in the retinal vein or the optic nerve
- A stroke or a brain injury
- Toxic substances: esp. quinine and methyl alcohol
- Others: high blood pressure, multiple sclerosis, serious vitamin deficiencies.

M C Q

1. the method of examination of anterior chamber angle is called:

- a) ophthalmoscopy
- b) Retinoscopy
- c) Gonioscopy
- d) Laparoscopy

Answer: (c)

2. Which statement of the following is wrong:

- a) Only central field is important in glaucoma.
- b) Peripheral field changes occur in glaucoma.
- c) Enlargement of blind spot is glaucomatous field change.
- d) Tubular field can occur in glaucoma.

Answer: (A)

3. Which of the following drugs does not decrease aqueous formation

- a) Beta blockers
- b) Prostaglandin analogues
- c) Carbonic anhydrase inhibitors
- d) Alpha adrenergic agonist

Answer: (B)

4. Neovascular glaucoma is least likely to occur as a result of:

- a) Central retinal vein occlusion
- b) Central retinal artery occlusion
- c) Branch retinal artery occlusion

- d) Proliferative diabetic retinopathy

Answer: (C)

5. Closed angle glaucoma can occur in predisposed eye with the use of:

- a) Atropine
- b) Cortisone
- c) Vitamin A
- d) Vitamin E

Answer: (A)

6. The mechanism of secondary glaucoma following central retinal vein occlusion is:

- a) Blood in trabecular meshwork
- b) Rise in IOP following rise in systemic blood pressure
- c) New vessel formation in the angle
- d) Angle recession

Answer: (C)

7. The best line of treatment for congenital glaucoma:

- a) Pilocarpine eye drops
- b) Beta blocker eye drops
- c) Peripheral iridectomy
- d) Goniotomy

Answer: (D)

8. Which of the following eye emergency can be misdiagnosed as acute abdomen:

- a) Acute iritis
- b) Acute angle closure glaucoma

- c) Acute conjunctivitis
- d) Central retinal artery occlusion

Answer: (B)

9. Glaucoma surgeries include all of the following except:

- a) Trabeculectomy
- b) Goniotomy
- c) Peripheral iridectomy
- d) Blepharoplasty

Answer: (D)

10.treatment of acute congestive glaucoma:

- a) Alpha adrenergic agonists eye drops
- b) Beta blocker eye drops
- c) Indentation gonioscopy
- d) All of the above

Answer: (C)

11.External fistulizing operations in glaucoma create channel bet:

- a) Posterior chamber and anterior chamber
- b) Posterior chamber and sub conjunctival space.
- c) Anterior chamber and sub conjunctival space.
- d) Posterior chamber and sub retinal space.

Answer:(C)

12.One of the following is not asymptom of acute congestive glaucoma:

- a) Rapid drop of vision.
- b) Severe itching.
- c) Severe bursting pain.
- d) Coloured haloes.

Answer:(B)

13.One of the following drugs can induced closed angle of glaucoma:

- a) pilocarpine nitrite
- b) atropine sulphate.
- c) prednisolone acetate.
- d) timolol maleate.

Answer: (B)

14.In absolute glaucoma the vision is HM:

- a). True b) False.

Answer:(B)

15.Causes of secondary glaucoma don't include:

- a) corneal ulcer.
- b) malure senile cataract.
- c) iridocyclitis.
- d) central retinal vein thrombosis.

Answer: (B)

16.Secondary glaucoma due to acute iridocyclitis should not be treated by:

- a) atropine 1%
- b) topical and systemic corticosteroids.
- c) pilocarpine.
- d) Diamox.

Answer: (C)

17.Which statement of the following is wrong about buphthalmos:

- a) can lead to perament damage to optic nerve.

- b) is always associated with small corneal diameter.
- c) its treatment is essentially surgical.
- d) can present with lacrimation.

Answer:(B)

18. Beta blockers are avoided when patient has:

- a) bronchial asthma.
- b) bladder problems.
- c) renal stones.
- d) allergy to apraclonidine

Answer: (A)

19. Neovascular glaucoma is most likely to occur as a result of:

- a) central retinal vein occlusion.
- b) b. central retinal artery occlusion.
- c) advanced retinitis pigmentosa
- d) proliferative diabetic retinopathy.

Answer: (C)

20. Which of the following could be considered as definitive treatment to neovascular glaucoma:

- a) Atropine eye drops.
- b) Steroid eye drops.
- c) Pan retinal photocoagulation.
- d) Peripheral iridotomy.

Answer:(C)

21. All of the following drugs can be used to treat glaucoma with uveitis except:

- a) timolol.
- b) pilocarpine.

- c) prostaglandin analogue.
- d) B+C

Answer:(D)

22.What is Glaucoma?

- a) Retinal damage form high IOP
- b) Optic nerve death caused by mechanical stretching forces
- c) Ischemic nerve damage from decreased blood transfusion
- d) None of the above

Answer: (B)

23.Aqueous humor is produced by:

- a) Cornea
- b) Lens
- c) Ciliary body
- d) Conjunctiva

Answer: (C)

24.Aqueous fluid is produced in which chamber?

- a) Anterior
- b) Posterior
- c) Vitreous
- d) Trabecular

Answer: (B)

25.Acetazolamide lowers IOP by:

- a) Decreased aqueous production
- b) increased aqueous drainage
- c) lower episcleral venous pressure
- d) All of the above

Answer: (A)

26. Which of the following is true concerning the IOP?

- a) Varies during the day with a peak in the early morning
- b) Normal value is 9-21 mmHg
- c) Can be normal in patients with glaucoma
- d) All of the above

Answer: (D)

27. Glaucoma inversus can occur in :

- a) Post subluxated lens.
- b) Post dislocated.
- c) Intumescent cataract.
- d) None of the above.

ANSWER: (C)

28. Glaucoma inversus can be treated by :

- a) Pilocarpine + anti-inflammatories.
- b) Pilocarpine + lens removal.
- c) Atropine.
- d) Cyclocryotherapy.

ANSWER: (C)

29. In buphthalmos we should exclude all of the following except :

- a) Retinoblastoma
- b) Megalocornea
- c) High myopia

- d) Babies of diabetic mothers

ANSWER: (D)

30. In buphthalmos which of the following is late presentation :

- a) Lacrimal and sneezing
- b) Optical cupping
- c) Enlarged hazy cornea
- d) Flattened subluxated lens

ANSWER: (B)

31. Acetazolamide lowers IOP by:

- a) Decreased aqueous production.
- b) Increased aqueous drainage.
- c) Lower episcleral venous pressure.
- d) All of the above.

ANSWER: (A)

32. B-blockers Lower IOP by:

- a) Decreased aqueous production.
- b) Increased aqueous drainage.
- c) Lower episcleral venous pressure.
- d) All of the above.

ANSWER: (A)

33.The eye susceptible to AACG:

- a) Hypermetropic eye.
- b) Myopic eye.
- c) Astigmatic eye.
- d) Aphakic eye.

ANSWER: (A)

34.In an acute angle closure glaucoma the choice of surgery is decided after:

- a) Gonioscopic examination.
- b) Fundus examination.
- c) Tonometry.
- d) Visual field examination.

ANSWER: (A)

35.Rapid painful loss of vision in all except:

- a) AACG.
- b) Blunt trauma.
- c) Alkali burn.
- d) CRAO.

ANSWER: (D)

36.In a patient with HM vision , visual field can be tested by :

- a) Projection of light

- b) Confrontation test
- c) Automated perimetry
- d) Bjerrum screen

ANSWER: (A)

37. Which of the following is not a test for visual field:

- a) Projection of light
- b) Confrontation test
- c) Automated perimetry
- d) Bjerrum screen
- e) Perception of light

ANSWER: (E)

38. All of the following are the characteristics of glaucomatous cup except:

- a) Large deep cup.
- b) Overhanging margins.
- c) Retinal vessels appear broken at the margin.
- d) Lamina criprosa is not visible.

ANSWER: (D)

Retina

ESSAY QUESTIONS:

1. What is the etiology and risk factors of central retinal vein occlusion?

Systemic predisposing factors:

1. Systemic hypertension.
2. Diabetes mellitus.
3. Blood disease: leukemia, polycythemia, sickle-cell disease (hyperviscosity syndromes).
4. Old age (6th & 7th decade).

Ocular predisposing factors:

1. Rise of IOP.
 2. Hypermetropia (small eye).
 3. Periphlebitis.
 4. Congenital anomalies in vein wall (strictures & tortuosity).
-

2. Describe the clinical picture (including fundus picture) of central retinal vein occlusion.

a) Symptoms:

Non ischemic CRVO:

- Moderate loss of vision, noticed more in the early hours of the morning following sleep. (During sleep, circulation is sluggish & venous pressure in the head increases).

Ischemic CRVO:

- Severe or marked loss of vision.

b) Signs:

Non-ischemic central retinal vein occlusion:

- Mild RAPD.
- Fundus picture (ophthalmoscopy):

Ophthalmoscopy shows:

- a. Mild tortuosity and dilatation of all branches of the central retinal vein.
- b. Mild retinal edema (macular).
- c. Mild retinal hemorrhages, flame-shaped or punctate (dot-and-blot), scattered at the central fundus and at the periphery in all quadrants.
- d. Mild Disc edema.
- e. Few, if any, cotton wool spots.

Ischemic central retinal vein occlusion:

- Marked RAPD.
- **Ophthalmoscopy shows:**
 - a. Marked tortuosity of all branches of central retinal vein.
 - b. Extensive retinal & macular edema.
 - c. Extensive retinal hemorrhages
 - d. Many cotton wool spots indicating capillary occlusion (microinfarctions)
 - e. Extensive disc edema
 - f. Extensive edema at the macular area up to cystoid macular edema
 - g. Development of new vessels at the disc (NVDs) and elsewhere (NVEs)
 - h. In severe cases, rubeosis iridis (iris new vessels) and new vessels at the angle of the AC cause secondary glaucoma (neovascular or 100-day glaucoma).

3. Outline the lines of treatment of CRVO.

1. Control of hypertension and diabetes.
2. After resolution of hemorrhages, fluorescein angiography is performed to detect ischemia.
3. Antiplatelet medication like aspirin 75 mg/day.
4. Argon laser photocoagulation in ischemic cases and in cases with neovascular glaucoma, but only after hemorrhages resolve.
5. Intravitreal drug injection: Steroids and/or Antivascular Endothelial Growth Factors (AntiVEGFs) for edema and neovascularization.

6. Neovascular glaucoma: is difficult to control and may require:
 - a. Extensive pan retinal photocoagulation
 - b. Cyclocryotherapy
 - c. Insertion of a valve as Ahmed valve or Molteno tube

4. Give etiology, clinical picture, fate and treatment of CRAO.

Etiology:

Thrombosis following atherosclerosis, which is more prevalent in patients with diabetes and hypertension and in hypermetropic patients.

Embolism:

- Detached thrombus as in:
 - Myocardial infarction
 - Aortic aneurysms
 - Subacute bacterial endocarditis
 - Mural thrombus in cases of AF
- Air embolism.
- Fat embolism as in:
 - Crush injury
 - Acute hemorrhagic pancreatitis

Spasm of the central retinal artery as in:

- Hypertensive encephalopathy
- Migraine
- Quinine poisoning

Arteritis as in:

- Giant cell arteritis
- Polyarteritis nodosa

Raised IOP, if the pressure is very high as during retinal detachment surgery or after injection of expansible gases during vitrectomy procedures

*Clinical picture:**a) Symptoms:*

- Sudden painless loss of vision.

b) Signs:

1. Normal external appearance of the eye.
2. Pupil:
 - Dilated
 - with loss of direct light reflex (relative afferent pupillary defect or RAPD)
 - and preservation of the consensual
3. Severe impairment of visual acuity (up to no perception of light) except in patients with cilioretinal artery
4. Fundus picture:
 - a. Attenuated arteries & segmented veins
 - b. Extensive central retinal edema due to coagulative necrosis of inner retinal layers & cloudy swelling of ganglion cells.
 - c. Cherry red spot at macular area (contains only cones which receive blood from underlying choroid, retaining red color against rest of retina which appears milky white due to coagulative necrosis of ganglion cell layer).
 - d. Marked narrowing of retinal arterioles. Blood maybe dark due to stasis.
 - e. Optic atrophy is end stage.

c) Fate:

1. Death of the inner half of the retina supplied by the CRA occurs in 5-10 minutes. It's irreversible.
2. The central vision may sometimes be preserved due to the presence of a cilioretinal artery, arising from the posterior ciliary arteries, which supply the macular area in 15% of the population.
3. After few weeks the color of the retina returns to normal as the macrophages engulf the dead ganglion cells and the disc shows consecutive optic atrophy.

Treatment:

- Should start immediately.
- The aim of ttt is to lower the IOP to induce reflex vasodilation and dilate the artery to eliminate the obstruction and dislodge the embolus or thrombus.

- Prognosis is bad, but all patients should be treated as an emergency.
1. Patient should lie flat
 2. An attempt is made to dislodge the embolus into a more peripheral arterial branch by:
 - a. Ocular massage to lower the IOP
 - b. IV acetazolamide 500mg to lower IOP
 - c. Anterior chamber paracentesis
 3. Inhalation of a mixture of 5% Carbon dioxide and 95% oxygen to dilate the retinal vessels
 4. Streptokinase injection may be useful in the first 24 hours
 5. Search for the cause as it may be due to a life-threatening disease as cardiovascular disease or emboli

5. Diabetic retinopathy (types, fundus picture, complications & management).

Definition:

- Bilateral retinal affection due to diabetes mellitus.
- Diabetes mellitus is a systemic disease characterized by sustained hyperglycemia secondary to lack or diminished efficacy of endogenous insulin.

Types of DM:

- a. Type I (insulin-dependent).
- b. Type II (non insulin-dependent).

Risk factors of diabetic retinopathy:

1. Duration of diabetes:

50% of diabetic patients develop DR after 10 years & 90% after 30 years.

2. Metabolic control:

Delays (not prevents) the condition.

3. Miscellaneous factors:

- Pregnancy.
- Hypertension.
- Renal disease

- Smoking.
- Anemia.

Pathogenesis:

Diabetic retinopathy is microangiopathy affecting retinal capillaries, arterioles & venules in the form of:

1. Microvascular occlusion, due to:

a. Pathology in vessel wall:

- Thickening of basement membrane.
- Loss of pericytes.
- Endothelial cell proliferation.

b. Changes in blood characters:

- Lack of deformability of RBCs (due to glycosylation of hemoglobin).
- Increased stickiness and aggregation of platelets.
- These factors cause non-perfusion of capillaries, leading to ischemia & retinal hypoxia which initiate formation of arterio-venous shunts & neo-vascularization.

2. Microvascular leakage:

- Due to breakdown of blood-retinal barrier as a result of loss of pericytes.
- This leads to leakage of plasma constituents into the retina causing retinal edema (diffuse or localized).

Types of diabetic retinopathy:

1. Non-proliferative diabetic retinopathy (NPDR):

Main findings are:

- Microaneurysms: showed both clinically & by fluorescein angiography (FA).
- Deep hemorrhages (blots and dots).
- Hard exudates.
- Retinal edema: if close to macular area, vision is seriously affected. In advanced cases, cystoid macular edema will develop.

2. Pre-proliferative diabetic retinopathy:

Due to retinal ischemia, shows the following:

- Venous changes: beading & looping.

- b. Hemorrhages.
- c. Cotton wool spots (capillary occlusion).
- d. Intraretinal microvascular abnormalities (IRMA).
- e. Extensive areas of ischemia (detected by FA).

3. *Proliferative diabetic retinopathy (PDR):*

Affects type I more than type II.

As retinal ischemia increases, ischemic retina secretes vasostimulating substances that result in budding of new vessels:

- a. Neovascularization at the optic disc (NVDs) or along course of retinal vessels, new vessels elsewhere (NVEs). They are unhealthy vessels that are liable to leak or rupture leading to repeated hemorrhages.
- b. Vitreous detachment.
- c. Hemorrhage maybe retinal, pre-retinal (subhyaloid) or vitreous.
- d. Later, fibrosis occurs around new vessels leading to traction on retina (tractional retinal detachment (retinitis proliferans)).

4. *Diabetic maculopathy:*

Definition:

- Presence of any retinal thickening or hard exudates within 1500 micrometer of the center of fovea. If edema or exudates are 500 micrometer from fovea, it's called clinically significant macular edema.
- Involvement of fovea by edema and/or hard exudates is the most common cause of visual impairment in diabetic patients.

Complications of DR:

Occur if laser treatment was not implemented in early stages, they include:

1. Persistent vitreous hemorrhage.
2. Retinal detachment.
3. Opaque membranes.
4. Rubeosis iridis, which may lead to neovascular glaucoma.

Management of DR:

1. Follow-up of diabetic patients and regular fundus examination every year.
2. If retinopathy develops, fluorescein angiography (FA) is done to detect leaking areas, with

follow up every 6 months.

3. Leaking microaneurysms causing macular edema are managed by focal argon laser photocoagulation.
4. If patient develops new vessels (NVDs or NVEs or rubeosis iridis), pan retinal photocoagulation is indicated using argon or diode laser.
5. Vitrectomy, in the following cases:
 - Recurrent vitreous hemorrhage.
 - Tractional retinal detachment.
 - Rubeosis iridis associated with vitreous hemorrhage (+ intra-operative pan laser photocoagulation to prevent neovascular glaucoma).

6. Ocular manifestations of diabetes mellitus.

1. *Lids:*

- a. Recurrent sty.
- b. Xanthelasma.
- c. Blepharitis.

2. *Cornea:*

Recurrent corneal erosions.

3. *Iris:*

- a. Rubeosis iridis: better seen at pupillary border.
- b. Diabetic iritis.

4. *Retina:*

Diabetic retinopathy.

5. *Lens:*

- a. True diabetic cataract.
- b. Presenile cataract.
- c. Hyperglycemia causes myopia.
- d. Hypoglycemia causes hypermetropia.

6. *Optic nerve:*

Ischemic optic neuropathy.

7. *Paralytic squint:*

Commonest muscle affected is lateral rectus.

7. Hypertensive retinopathy.

Pathogenesis:

Retinal arterioles respond to hypertension by narrowing.

Fundus picture:

1. Vasoconstriction.
2. Leakage due to abnormal permeability of vessel wall, leading to flame shaped hemorrhages, edema & hard exudates.
3. Disc edema may develop in malignant hypertension.
4. Arteriosclerosis causes thickening of vessel walls.
5. Arteriovenous crossing changes (most important finding, mentioned below).

Grading:

Grade 1:

Mild generalized arteriolar attenuation, esp. small branches.

Grade 2:

1. More severe, generalized & focal arteriolar constriction.
2. Deflection of veins at arteriovenous crossings (Salus' sign).

Grade 3:

1. Copper wiring of arterioles.
2. Banking of veins distal to A/V crossing changes.
3. Tapering of veins on either side of the crossings.
4. Flame shaped hemorrhages.
5. Cotton wool spots.
6. Hard exudates.

Grade 4 (malignant hypertension):

All of the above + silver wiring of arterioles& disc edema.

*Other ocular manifestations of hypertension:*Plays a role in:

1. Branch retinal vein occlusion.
2. Retinal artery occlusion.
3. Ischemic optic neuropathy.
4. Ocular motor nerve palsy.
5. Diabetic retinopathy.

8. Retinopathy of malignant hypertension & renal failure.

= Grade 4.

- Occurs on top of benign hypertension.
- Vessels are attenuated and straight if develops rapidly.
- Hemorrhage of all types (flame shaped, dot & blot).
- Cotton wool spots (micro-infarctions, numerous).
- Exudate may radiate from fovea along fiber layer forming macular fan or star.
- Disc edema is common.
- Sometimes diffuse retinal edema (esp. in renal cases), causing exudative RD.
- Death from renal failure, unless treated.

9. Retinopathy of toxemia of pregnancy.*Definition:*

Bilateral retinal affection secondary to toxemia of pregnancy.

Pathology:

- Affects females after 20th week of pregnancy.
- Due to effect of hypertension in toxemia of pregnancy, retinal arterioles show marked vasoconstriction leading to ischemia.

*Clinical picture:**Symptoms:*

Mild cases show no symptoms. Severe cases show:

1. Metamorphopsia and blurring of vision due to macular exudates and hemorrhage.
2. Rapid painless diminution of vision due to exudative RD.

Signs:

1. Spasm and narrowed retinal arterioles.
2. Flame-shaped hemorrhage.
3. Cotton wool exudates due to microinfarctions.
4. Papilledema.

Complications:

1. Macular edema and exudates causing diminution of vision.
2. Exudative retinal detachment, characterized by:
 - Rapid onset.
 - Bilaterality.
 - Absence of retinal breaks.
 - Convex, stretched retinal configuration.
 - Shifting of subretinal fluid.

NB.

- Termination of pregnancy is indicated if patient develops exudative RD with rapid bilateral onset in absence of retinal breaks.
- Retinopathy resolves after termination of pregnancy with good vision prognosis.

10. Age-related macular degeneration.

The leading cause of irreversible severe visual loss above 60 years.

Types:

1. Dry AMD.
2. Wet AMD.

1. Dry AMD:

- 90% of cases.

- Results from slowly progressive atrophy of RPE and photoreceptors.

Clinical picture:

- Focal hyperpigmentation.
- Circular areas of RPE atrophy through which large choroidal vessels are seen.

2. Wet AMD:

- Rapid diminution of vision + metamorphopsia.

Signs:

- Choroid neovascular membrane.
- Macular edema, submacular hemorrhage & macular scarring.

Treatment:

1. Laser photocoagulation.
 2. Photodynamic therapy (PDT): A dye is injected IV and the lesion is exposed to a laser of a certain wave length to produce a chemical reaction that coagulates the neo-vessels.
 3. Intravitreal injection of anti-VEGFs (standard ttt).
- Poor prognosis.

11. Anti vascular endothelial growth factors (anti VEGFs).

- Vascular endothelial growth factors are responsible for growth of new blood vessels by stimulating the endothelial cells which form the walls of the vessels and transport nutrients and oxygen to tissues.
- Neovascularization occurs when retinal pigment epithelial cells experience lack of nutrition (ischemia).
- VEGFs also act to restore function in other parts of the body.
- However, these new vessels are not formed properly & leak. This leakage results in macular scarring & eventual loss of central vision.
- Anti-VEGFs prevent VEGFs from binding to their receptors on surface of endothelial cells. They are injected intravitreal.
- Examples include: Avastin.
- Uses:
 1. Age related macular degeneration.

2. Diabetic macular edema.
3. Central & branch vein occlusion leading to macular edema.

12. Rhegmatogenous (primary) retinal detachment

Definition:

Separation of the neurosensory retina from the RPE by subretinal fluid due to the presence of retinal break.

Etiology:

Causes of vitreous liquefaction (risk factors) include:

1. High axial myopia
2. Aphakia especially by ICCE
3. Trauma
4. Family history of RD
5. RD in the other eye

Retinal break:

Definition:

Full thickness defect in the neurosensory retina.

Morphological types:

Tear:

Due to vitreoretinal traction at sites of vitreoretinal adhesions (horse shoe or arrow heads, usually upper especially temporal)

Giant tear:

If it affects 90 degrees or more of the circumference

Dialysis (or disinsertion):

Due to circumferential disinsertion of the retina from the ciliary body at the ora serrata, usually traumatic and upper nasal

Hole:

Less risk as no vitreoretinal traction

Rounded or oval due to retinal atrophy or degeneration e.g. myopic lattice

degeneration

Usually temporal and upper at the ora serrate especially in aphakics

Operculated hole:

In which the flap is completely torn and separated

Site of breaks:

1. Oral : at vitreous base; ora serrate
2. Post-oral: between vitreous base and equator
3. Equatorial
4. Post-equatorial: posterior to equator
5. Macular (foveal): usually holes

Clinical picture:

Symptoms:

1. Due to retinal break:

- a. Flashes (photopsia): due to separation of the photoreceptors
- b. Floaters (musca volitans) in the form of a sudden shower of minute red colored or dark spots due to vitreous hemorrhage from tearing of the blood vessels
- c. Drop of vision (rapid painless), rare, if the vitreous hemorrhage was severe

N.B.

- Flashes (photopsia) may occur from PVD (posterior vitreous detachment) even in the absence of a break
- Floaters may be due to an operculated break where the floating flap in the vitreous cast a shadow on the retina

2. Due to retinal detachment:

- a. Field defect: described as a black curtain or veil, due to spread of the subretinal fluid
- b. Drop of central vision: if the macula is involved by the detachment (involvement of the fovea by subretinal fluid of foveal detachment)

N.B.

- The quadrant in which RD appears first predicts the location of the 1st break (opposite to it)

Signs:

Vision: dropped if macula affected

Pupil: relative afferent pupillary defect (Marcus-Gunn pupil), if extensive or total

Fundus:

Red reflex: grayish at the site of RD due to the turbid subretinal fluid

The break: appears dark red due to visibility of the underlying choroid

Fresh or early, the detached retina appears:

- Grayish, elevated and folded with tortuous blood vessels
- Convex wavy configuration with a corrugated thickened surface (due to intra-retinal edema)
- Mobile

Late signs in long standing cases:

- Proliferative vitreoretinopathy (PVR): due to metaplasia and proliferation of the RPE cells with secondary migration in the inner retinal surface (epiretinal membranes) or occasionally outer retinal surface (subretinal membranes) leading to fixed retinal folds with rigidity of the retina (less mobile), it's the commonest cause of recurrence and failure of surgery if not removed completely
- Retinal thinning (atrophy)
- Subretinal pigmented demarcation lines or water marks due to RPE proliferation between detached and attached retina (3 months)
- Intraretinal cysts (one year)

Slit lamp examination (anterior segment signs):

- The anterior chamber might show mild uveitis (secondary uveitis).
- The anterior vitreous might show tobacco dusting (+ve Shaffer sign).
- In long standing cases:
 - Iris: rubeosis iridis
 - Complicated cataract

IOP:

Mild hypotony (lower by about 5 mmHg) due to absorption of the SRF by choroid.

Treatment:

1. Prophylactic sealing; break without detachment:

Sealing of the break with two rows of laser or single row of cryotherapy.

Idea:

Creating an inflammatory chorioretinal lesion which on scarring results in strong adhesion between the sensory retina and the RPE/choroid preventing the development of RD.

Indications:

- Flat retinal tears (without RD):

Especially large, upper, symptomatic (flashes), risk factors (aphakic, myopic, family history or other eye).

- Peripheral retinal degeneration predisposing to RD (e.g. lattice):

Especially in the presence of risk factor (family history, aphakia, myopia, other eye).

2. Retinal detachment :

a. Scleral buckle surgery: (conventional RD surgery)

Idea:

- Find and seal the break and oppose the retina to the underlying choroid.
- Drainage of subretinal fluid by the aid of sclerotomy if:
 - Long standing (viscous)
 - Large amount.
 - Atrophic choroid e.g. : high myopia
 - Sealing of the break.
- Application of a scleral buckle to indent the sclera at the site of the break by using an explant sutured to the sclera for:
 - Closure of the break by opposing the RPE to sensory retina.
 - Reduction of vitreo-retinal traction.

b. Pars planavitrectomy with silicone oil injection :

Indicated in:

- Breaks that are posterior (as macular hole) or giant or multiple at different sites.
- PVR.
- RD with vitreous hemorrhage.

NB:**3 ports:**

- Infusion port.
- Vitrectomy cutting probe.
- Illumination probe.

Complication of silicone oil include:

- Emulsification:

May be present with an inverted hypopyon. That's why a lower iridectomy is usually done if silicone oil is injected (Ando's or Japanese or 6 o'clock iridectomy).

- Band shaped keratopathy.
- Cataract.
- Glaucoma.

Silicone oil is removed after 6 months after securing that the retina is in place or if it leads to complications.

13. Retinal examination in RD.***1. Indirect ophthalmoscopy:***

- Extent of RD:
Breaks' sites, shape, size and number.
- State of macula.
- PVR (presence and severity).

NB: Fresh RD with macular attachment (macula on) is considered an ocular emergency that requires urgent surgery.

2. Slit-lamp bio-microscopy:

For a magnified detailed image of retina and vitreous but with a smaller field.

3. Ultrasonography (B-scan):

- In suspected RD in the presence of opaque media. e.g. cataract, corneal opacities and vitreous hemorrhage.
- To exclude intraocular tumor in exudative RD.

4. *Retinal function tests , esp. in long standing cases:*

- Color perception.
- Light projection.
- Electrophysiological tests (ERG, EOG, VEP).

14. **Enumerate types of RD.**

1. Rhematogenous (primary) RD.
2. Tractional RD.(secondary).
3. Exudative RD (secondary).

15. **Tractional retinal detachment.**

Etiology:

Retina is pulled by contracting glial tissue in the vitreous (vitreous-retinal traction).

1. Organized vitreous hemorrhage:
 - Proliferative diabetic retinopathy.
 - CRVO.
 - Trauma.
2. Cyclitic membrane.
3. Retinopathy of prematurity.
4. Posterior segment penetrating trauma.
5. Proliferative sickle retinopathy.

Clinical picture:

Symptoms:

1. No photopsia (vitreoretinal traction is gradual and not associated with acute posterior vitreous detachment).
2. No floaters.
3. Field defect: slowly progressive, even stationery (months).

Signs:

1. Detached retina appears:
 - No retinal breaks.
 - Concave, with highest elevation at site of traction.
 - Shallow (minimal or absent) subretinal fluid.
 - Restricted mobility.
 - No shifting fluid.
2. If traction results in a break, it assumes the characters of rhegmatogenous RD and progresses rapidly (combined tractional & rhegmatogenous RD).

Treatment:

Vitrectomy & silicone oil injection.

16. Exudative retinal detachment.

Etiology:

- Retina is pushed by choroidal lesions. The sub-retinal fluid derived from the choroid gains access to sub-retinal space through damaged retinal pigment epithelium (RPE).
- The least common type of RD.

Systemic causes:

1. Pregnancy induced hypertension.
2. Severe hypertension.
3. Hypoproteinemia.

Ocular causes:

1. Choroidal tumors as melanoma, hemangioma & metastasis.
2. Inflammations (choroiditis) as Harada & posterior scleritis.
3. Iatrogenic: extensive RD surgery.
4. Extensive leakage from choroidal neovascular membrane.

Clinical picture:

Symptoms:

1. No photopsia (no vitreoretinal traction).
2. No floaters (unless associated with vitritis).

3. Field defect: sudden & rapidly progressive.

Signs:

1. Detached retina appears:
 - No breaks.
 - Convex with smooth non corrugated surface.
2. Signs of the cause (e.g. choroidal tumor).

N.B. B scan ultrasonography could be done to diagnose the case (e.g. choroidal tumor).

Treatment:

Treat the cause.

17. Retinitis pigmentosa.

Definition:

Bilateral progressive symmetrical photo receptor and RPE dystrophy (abiotrophy) predominantly and initially affecting the rods with subsequent degeneration of the cones (rod-cone dystrophy).

Inheritance:

- Autosomal dominant: most common.
- X-linked recessive: most severe.
- Autosomal recessive.
- Sporadic.

Clinical picture:

Symptoms:

1. Night blindness (nyctalopia).
2. Progressive contraction of the field.
3. Defective vision and color (later on).

Signs:

1. Arteriolar attenuation.
2. Bone corpuscle (or spicule or spider like) pigmentation starting at the equatorial midperiphery followed by the periphery then the central retina (due to RPE migration towards the inner retinal layers along small arterioles and venules).

3. Color of the disc : waxy yellow.

Investigations:

1. *Electroretinogram (ERG):*

- Decreased scotopic response .
- Then photopic .
- Later on extinguished .

NB: decreased in females carriers in X-linked cases .

2. *Field:*

- Ring or annular scotoma.
- DD: double arcuate scotoma.
- Progressive contraction.
- Tubular field.

3. *Electro-oculo-gram (EOG)*

- Subnormal (due to RPE dysfunction) .

Ocular associations and complications:

1. Anterior segment:

- a. Complicated cataract.
- b. PAOG.
- c. Keratoconus.

2. Posterior segment:

- a. High myopia.
- b. Consecutive optic atrophy.
- c. Maculopathy (atrophic, cystoid, cellophane).
- d. PVD.

Systemic associations:

1. Refsum disease:

- Due to increased phytanic acid.
- Characterized by: deafness, ataxia, polyneuropathy, cardiac arrhythmias & salt and pepper retinopathy.

2. Bardet-Biedel syndrome:

Polydactyly, obesity, hypogonadism, mental retardation, bull's eye & maculopathy.

3. Usher syndrome:

Deafness, vestibular dysfunction & pigmentary retinopathy.

Treatment:

1. Low visual aids, e.g. night telescopes.
2. Treatment of complications, e.g. cataract-carbonic anhydrase inhibitors for macular edema.
3. Genetic counseling.
4. Recently: stem cells and gene therapy.

N.B. Vitamin A may decrease progression.

*D. D.*1. Decreased night vision:

- o Vitamin A deficiency.
- o High axial myopia.
- o Incipient cataract.
- o PAOG.

2. Tubular field:

- o PAOG.
- o Advanced pigmentosa.
- o Advanced papilledema.
- o CRAO with patent cilio-retinal artery.

3. Secondary or pseudo RP:

- o Siderosis bulbi (retained iron IOFB).

18.State the causes of defective night vision & describe clinical picture of the fundus diseases leading to night blindness.

Causes:

1. Vit A deficiency

2. Congenital night blindness
3. Retinitis pigmentosa
4. Advanced stage of chronic glaucoma
5. Cortical cataract

Clinical picture of fundus diseases "Retinitis Pigmentosa":

Symptoms:

- Night blindness
- Progressive contraction of field
- Defective vision & color

Signs:

- Arterioles → attenuation
- Bone spider specules → at mid periphery followed by periphery then central
- Colour of disc → waxy yellow

MCQ:

1. Symptoms of rhegmatogenous retinal detachment don't include:

- a. Headache.
- b. Photopsia.
- c. Musca volitans.
- d. Diminution of vision.

2. Retinitis pigmentosa is characterized by:

- a. Pigmentary degeneration of the retina.
- b. Chronic inflammation of the retina.
- c. Single focus of retinal pigmentation.
- d. Secondary optic atrophy.

- e. Unknown mode of inheritance.

3. Simple retinal detachment is characterized by the following except:

- a. Low intraocular pressure.
 - b. Opaque to trans-illumination.
 - c. Presence of retinal tear.
 - d. Eye is usually myopic.
-

4. Retinal detachment can be caused by:

- a. Retinopathy of the toxemia of pregnancy.
 - b. Non-proliferative diabetic retinopathy.
 - c. Central retinal artery occlusion.
 - d. Neglected primary open angle glaucoma.
-

5. In retinal detachment, the following are true except:

- a. Red reflex is always grey whatever the size of the pupil or the position of gaze.
 - b. In rhegmatogenous RD, retina is corrugated, mobile with wavy vessels.
 - c. Retina is anteriorly convex in exudative RD.
 - d. IOP maybe elevated.
-

6. Symptoms of retinal tears include:

- a. Flashes of light.
 - b. Ocular pain.
 - c. Headache referred to temporal bone.
 - d. Transient attack – loss of vision.
 - e. Nausea and vomiting due to vagal stimulation.
-

7. The earliest symptoms of retinitis pigmentosa:

- a. Defective night vision.
 - b. Delayed dark adaption.
 - c. Severely diminished reading ability.
 - d. Inability to move due to loss of peripheral field.
 - e. Slight ciliary injection.
-

8. Retinal detachment is:

- a. Separation between the external limiting membrane and the outer nuclear layer.
 - b. Separation between the outer nuclear and outer plexiform layers.
 - c. Separation between the RPE and neurosensory retina.
 - d. Separation between the outer plexiform layer and inner nuclear layer.
-

9. The risk factors for development of rhegmatogenous retinal detachment include the following except:

- a. Aphakia.
 - b. Hypermetropia.
 - c. Trauma.
 - d. History of retinal detachment of the other eye.
-

10. PVR is the proliferation of membranes on:

- a. Retina.
 - b. Cornea.
 - c. Conjunctiva.
 - d. Pupil.
-

11. Central retinal vein occlusion is characterized by all of these, EXCEPT:

- a. Retinal veins are dilated and tortuous.
 - b. Retinal hemorrhages.
 - c. Optic disc edema.
 - d. Cherry red spots.
-

12. In some patients with central retinal artery occlusion the central visual field may be spared, this may be explained by:

- a. Patent retinal veins.
 - b. Patent retinal arteries.
 - c. Patent cilioretinal artery.
 - d. Patent external carotid artery.
-

13. Central retinal vein thrombosis manifestations do NOT include:

- a. Retinal veins are dilated and tortuous.
 - b. Severe pain and headache.
 - c. Superficial hemorrhage all over the fundus.
 - d. Impairment of vision.
 - e. Secondary rubeotic glaucoma.
-

14. The risk factors in CRVO includes:

- a. Age over 50 years.
 - b. Hypertension.
 - c. Diabetes.
 - d. All of the above.
-

15. The patient with CRVO will need panretinal photocoagulation if:

- a. Angiogram demonstrates retinal non-perfusion areas.

- b. Macula develops cystoid macular edema.
 - c. Iris neovascularization develops.
 - d. There is relative afferent pupillary defect.
-

16. Neovascular glaucoma is least likely to occur as a result of:

- a. CRVO.
 - b. CRAO.
 - c. Branch retinal artery occlusion.
 - d. Proliferative diabetic retinopathy.
-

17. Mechanism of secondary glaucoma following CRVO is:

- a. Blood in trabecular meshwork.
 - b. Rise in IOP following rise in systemic blood pressure.
 - c. New vessel formation in the angle.
 - d. Angle recession.
-

18. Cherry red spot can be found in the following condition:

- a. Retinitis pigmentosa.
 - b. CRAO.
 - c. Central chorioretinitis.
 - d. Penetrating eye injury.
-

19. Causes of marked drop of vision in patients suffering from diabetic retinopathy is due to all of the following, except:

- a. Diabetic macular edema.
- b. Tractional retinal detachment affecting the macula.
- c. Vitreous hemorrhage.

- d. Retinal drusens.
-

20. Retinal endings of non proliferative diabetic retinopathy include:

- a. Cotton wool exudates.
 - b. Hard exudates.
 - c. Sub-hyaloid hemorrhage.
 - d. Flame-shaped hemorrhage.
-

21. Simple RD is characterized by the following except:

- a. Low IOP.
 - b. High IOP.
 - c. Presence of retinal tears.
 - d. Eye is usually myopic.
 - e. History of trauma.
-

22. In RD, following are true except:

- a. Red reflex is always grey whatever pupillary size and position of gaze.
 - b. In simple RD, retina is corrugated, mobile with wavy vessels.
 - c. Retina is anteriorly convex in exudative RD.
 - d. IOP may be elevated.
-

23. Laser photocoagulation is indicated in all of the following except:

- a. Diabetic retinopathy.
- b. Diabetic maculopathy.
- c. Rhegmatogenous RD.
- d. Retinal break with no sub retinal fluid.

24. Which of the following is risk factor for RD:

- a. Black race.
 - b. Male sex.
 - c. Presbyopia.
 - d. Myopia.
-

25. The commonest cause for night blindness:

- a. Congenital.
 - b. Vitamin A deficiency.
 - c. Nuclear cataract.
 - d. Retinitis pigmentosa.
 - e. Liver diseases.
-

Optic nerve

GIVE AN ACCOUNT

I. Write a short note about papillitis: (Type of optic neuritis)***Etiology:*****A. Non-infective:**

- 1) Demyelination disease e.g multiple or disseminated sclerosis (MS or DC) (the commonest). Young adult or middle aged female.
- 2) Toxic effect of drugs e.g anti-TB.
- 3) Nutritional e.g Vit. B deficiency, anemia.

B. Infective:

- 1) Causative organism:
 - I. Viral e.g influenza, measles (usually presents with a bilateral papillitis in children).
 - II. Bacterial e.g Syphilis, T.B.
- 2) Source: Extension from, Sinusitis, orbital cellulitis, uveitis, brain meningitis.

Mechanism:

Exudates (active edema). Usually unilateral.....bilateral if viral.

Clinical Picture:

Symptoms:

1. Drop of vision (rapid, Marked).
2. The colors appear to be washed out.
3. No pain in papillitis.

Signs:

- Vision: dropped.
- Color: Red-Green Dyschromatopsia out of proportion of VA.
- Pupil: afferent defect (marcus Gunn Pupil), sluggish direct and intact indirect.
- fundus:
 - a. Hyperemia of the disc.
 - b. Disc Swelling or disc edema (exudation)
 - ill-defined disc margin.
 - Cup obliteration.
 - Elevated (not more than + 3D = 1mm)
 - c. Vitreous inflammatory cells: Turbid dust like opacities.
 - d. vessels are tortuous and leaky.

Investigations:

- Field:

Relative negative central or centioceacalscotoma for red and green.
- Visual evoked cortical potential:

Increased latency and decreased amplitude.
- Of Cause, MRI brain:

to exclude MS (demyelinating plaques)

Complications (fate):

- 1) Recovery within weeks or months.

- 2) Recurrence is common in MS.
- 3) Optic atrophy: Secondary in papillitis.

Treatment:

Of the cause: send to neurologist if suspected MS for systemic steroids +/- interferon.

Aim→ Decrease the risk and severity of MS and speed up the recovery.

Dose→ Pulse therapy of 1 gm IV methylprednisolone (250 mg/6 hours) followed by oral full dose prednisolone 1 mg/Kg/d for 11 days (+/- interferon as stated by neurologist) to be followed by a slow gradual withdrawal over 4 days.

N.B: Retrobulbar neuritis:

- Pain → with ocular movement (esp. in and up) because medial rectus and superior rectus are attached to the ON sheath.
- Fundus → Normal.
- Optic atrophy → Primary.
- Causes → Multiple sclerosis-usually unilateral.

2. Discuss papilledema:

Definition:

Is a **bilateral** passive non inflammatory swelling of the optic nerve head (disc edema) due to increased intracranial tension

Mechanism:

It occurs due to block of axoplasmic flow resulting in swelling of the axons that causes venous congestion and extracellular fluid accumulation (**transudate**).

Etiology: (SBSB)

- 1) Space occupying lesion: e.g intra cranial tumors:
 - a) 70% of papilledema Cases.
 - b) bilateral unless one nerve is atrophic.
foster-kennedy syndrome: frontal lobe tumor presenting with ipsilateral primary optic atrophy and a contralateral papilledema.
- 2) 2. Benign increased tension (Idiopathic intracranial hypertension)
 - a) Young obese females on OCPs.
 - b) Hypervitaminosis A & D.

- c) Endocrine disorders e.g Addison's and Cushing.
- 3) Vascular: Subarachnoid hemorrhage.
- 4) Blockage of the CSF Circulation:
 - a) Congenital aqueductal stenosis.
 - b) Inflammatory e.g Meningitis, Tuberculoma.

Clinical picture:

Symptoms:

- a. General symptoms of increased intracranial tension:
e.g Headache, projectile vomiting (not preceded by nausea).
- b. Ocular symptoms:
 - 1. No symptoms.
 - 2. Diplopia at 6th Nerve palsy +/- bilateral (as a false localizing sign to ICT)
 - 3. Amaurosisjugax usually postural (due to sudden block of transmission or spasm of central retinal artery).
 - 4. Drop of vision (gradual and painless): **late** due to progressive optic atrophy.
- c. Of the cause.

Signs:

- 1. Vision:
 - not affected early, late gradual drop of vision due to:
 - loose arrangement of fibers in lamina cribrosa that allows much accommodation of fluid before compressing optic nerve fibers.
 - last fibers affected are the PMB.
- 2. Pupil:
 - not affected early, later an afferent defect esp. in symmetrical cases.
- 3. Fundus:
 - Early:
 - blurring of **nasal** margin.
 - loss of spontaneous venous pulsations (if they were present even with pressure on globe).
 - Established:
 - blurring of all disc margin (swelling up to +9D = 3 mm by direct ophthalmoscopy)

- obliteration of the physiological cup.
- Dilated tortuous veins.
- leakage around the disc (peripapillary) edema, flame, shaped hges, hard exudates.
- the exudates may extend to the macula alonghenle fiber layer giving a macular fan (incomplete star) configuration.
- enlarged blind spot.
- Chronic:
 - Elevated gliotic resembling the champagne cork or mushroom appearance.
 - tubular field.
- Atrophic:
 - post-papillaedemic secondary optic atrophy.
 - sheathing by gliosis.

Investigations:

1. Field of Vision:
 - a. Early (established stage) enlarged blind spot due to seperation of photo-receptor around the disc by edema.
 - b. progressive contraction of the field up to tubular field.
2. Of cause:
 - neuro-imaging (CT, MRI, brain) to exclude brain tumor.
3. Others:
 - colour vision: central scotoma for blue.
 - VEP: normal early.
 - FA: disc leakage.

Complications:

Secondary post-papilledema optic atrophy.

Treatment:

1. Send to neurologist to treat the cause.
2. In idiopathic intracranial hypertension.
 - a. Stoppills and reduce weight.
 - b. Surgical:
 - I. Optic nerve sheath decompressed if progressive visual loss as detected by regular field examination
 - II. Shunt surgery if severeheadache.

D.D. ➔: Papillitis and high axial hypermetropia (pseudo-papilledema or pseudo-neuritis).

3. Differentiate in a table bet. Papillitis and papilledema:

See above.

Don't forget to focus on (mechanism, laterality, vision, pupil, disc swelling, hyperemic disc, vessels, vitreous, field, ...)

4. Discuss Anterior ischemic optic neuropathy:

Definition:

Partial or total infarction of the optic nerve due to occlusion of the short posterior ciliary arteries.

Types: Arteritic and non arteritic.

A. Arteritic ischemic optic neuropathy:

Age: more serious type affecting mainly older patients (>70)

Causes: suffering from giant cell or temporal arteritis.

- Autoimmune occlusive vasculitis affecting the media and adventitia of large and medium sized arteries esp. superficial temporal, short posterior ciliaries, vertebral, ophthalmic.

Symptoms:

- 1) Rapid painful severe drop of vision (upto no PL).
- 2) General symptoms of GCA e.g. headache, weight loss, polymyalgia rheumatic, jaw claudication, scalp tenderness, night fever.

Signs:

- 1) vision: upto no PL
- 2) colour : dropped
- 3) pupil: RAPD
- 4) fundus: Pale swollen disc (chalky white) with splinter hge.

Complication:

Optic atrophy (+/- cupped)

Prognosis is poor

Management:

1) **Emergency**

As 65% of untreated patients become bilaterally blind within few days or weeks (bilateral simultaneous affection is rare).

- 2) The main of treatment is to prevent the other eye affection.
- 3) Urgent ESR (may reach above 60mm), C-reactive ptns and temporal artery biopsy must be done to confirm diagnosis.
- 4) Intensive systemic steroids should be initiated immediately.

B. Non arteritic ischemic optic neuropathy:

Age: Commonly in patients bet. 45 and 60 years.

Risk factors:

- 1) Hypertension, atherosclerosis and diabetic.
- 2) Others: Smoking, hyperlipidemia and sleep apnea syndrome.

Symptoms:

Drop of vision (Rapid, painless, moderate to severe)

Signs:

- 1) Vision: dropped
- 2) Colour: impaired in proportion to visual acuity (DD: optic neuritis)
- 3) Pupil: Marcus Gunn pupil
- 4) Fundus: Sectorial or diffuse pale disc edema =/- splinter shaped he.

Investigation:

- 1) Field:
 - Altitudinal field defect (usually inferior) due to segmental occlusion of SPCA.
 - +/- arcuate or central if affects PMB.
- 2) Of the cause
- 3) If in doubt; exclude arteritic type (ESR, CRP, TAB).

Complication: → *secondary optic atrophy*

Treatment:

- 1) Of the cause (BP, smoking,)
- 2) Low dose aspirin
- 3) Avoid the antihypertensive treatment before night.

5. Discuss the different types of optic atrophy:

- Optic atrophy: degeneration of the optic nerve fibers due to interruption of the nerve fibers at any point between the retinal ganglion cells and the lateral geniculate body.

Clinical picture:

- Vision: No PL.
- Colour: absent.
- Pupil: RAPD in unilateral or asymmetrical cases
- Field: no field.
- Fundus: according to type:

- **Primary:**

- **Causes:**

1. Inflammatory:
 - retro-bulbar neuritis (DS)
 - Tabes dorsalis (Syphilis)
2. Compressive:
 - Tumours compressing on ON. Chiasma and tract.
3. Traumatic: fracture base.
4. Ischemic: Severe blood loss.
5. Hereditary.

- **Color: Milky white.**

- **Edge:** Well-defined.

- **Cup:** Saucerized slightly enlarged.

- **Lamina:** Seen.

- **Vessels:** Normal.

Neurologist/ MRI/ CT.

- **Secondary:**

- **Causes:**

1. Post papillitis.
2. Post papilledema.

- **Colour:** Grey or dirty yellowish white.

- **Edge:** Ill-defined.

- **Cup:** Obliterated.

- **Lamina:** not seen.
- **Vessels:** Sheathed attenuated.

Elevation.

- ***Consecutive:***

- **Causes:**

1. CRAO.
2. Retinitis pigmentosa.
3. Retinal detachment.
4. High degenerative myopia.

- **Colour:** Waxy yellow.
- **Edge:** Ill-defined.
- **Cup:** Obliterated.
- **Lamina:** not seen.
- **Vessels:** Sheathed attenuated

Retinal disease.

- ***Glaucomatous:***

- **Causes:** Absolute glaucoma.
- **Colour:** white.
- **Edge:** Well-defined overhanging (undermined).
- **Cup:** Wide and deep.
- **Lamina:** seen +/- stippled.
- **Vessels:** Nasal shift, kinking limbing.

High IOP.

Treatment:

Of the cause +/- Vit. B complex, prognosis is poor.

MCQS:

1. Alcohol amblyopia is:

- a) Reversible and carry good prognosis.
- b) Due to ingestion of ethyl alcohol.
- c) Due to ingestion of methyl alcohol
- d) Result in cherry red spot.

Answer: (C) Due to ingestion of methyl alcohol

2. Retiobulbar neuritis is characterized by:

- a) May be acute or chronic.
- b) Shows central or paracentral scotoma forced and green.
- c) Inflammation of optic nerve behind the eye.
- d) May be due to disseminated sclerosis.
- e) All of the above.

Answer: (E) All of the above.

3. Primary optic atrophy is characterized by:

- a) Optic disc is white in color.
- b) lamina cribrosis is well seen.
- c) Retina and blood vessels are normal.
- d) All of the above.

Answer: (D) All of the above.

4. Optic nerve head in glaucomatous optic atrophy has all except:

- a) Large deep cup.
- b) Interrupted retinal vessels.
- c) Waxy yellow colour.
- d) Overhanging margins.

Answer: (C)

5. Doctor sees nothing & patient see nothing is:

- a) Papillitis.
- b) Papilloedema.
- c) Retrobulbar neuritis (toxic amblyopia).
- d) All of the above.

Answer: (C)

6. Which of the following ttt is used for optic neuritis:

- a) Prednisolone.
- b) Observation.
- c) Antibiotics.
- d) Atropine.

Answer: (A)

7. Papilloedema leads to:

- a) Rapid deterioration of vision.
- b) Primary optic atrophy.
- c) Pain on eye movements.
- d) Optic disc edema more than 3D.

Answer: (A)

8. Papilloedema leads to the following changes:

- a) Nasal step.
- b) Arcuate scotoma.
- c) Concentric contraction of peripheral field.
- d) Enlarged blind spot.

Answer: (D)

9. Consecutive optic atrophy occurs in all except:

- a) Degenerative myopia.
- b) Chorioretinitis.

- c) CRAO.
- d) CRV thrombosis.

Answer: (D)

10. Afferent pupillary defect occurs in all except:

- a) Papillitis.
- b) Hysteria.
- c) Optic atrophy.
- d) Retrobulbar neuritis.

Answer: (B)

Neuro-ophthalmology

COMPARE BETWEEN: (V.V.V.IMP.):

	<i>Miosis</i>	<i>Mydriasis</i>
<i>Physiological:</i>	• Sleep. • 3rd stage of anaesthesia. • Senile-neonate. • Light & near reflex. • Corneo-pupillary reflex.	• Excitement • 2nd stage of anaesthesia. • Light coloured iris. • Light withdrawal. • Cilio-spinal reflex.
<i>Drugs:</i>	• Miotics (pilocarpine). • Opium (morphine).	• Mydriatics (atropine). • Datura.
<i>Ocular:</i>	• Acute iridocyclitis • Trauma • Paracentesis & hypotony.	• Acute congestive glaucoma. • Trauma.
<i>Neurological:</i>	• Horner • Argyll-Robertson pupil. • Pontine hge. • Cerebral compression. (irritative stage).	• 3rd nerve palsy (esp. surgical causes e.g: PCAA). • Adies pupil. • 4th stage anaesthesia. • Cerebral compression (paralytic).

GIVE AN ACCOUNT

1. Enumerate the abnormal pupillary responses:

- 1) Afferent pupillary defect as Marcus Gunn (relative) and Amaurotic (total).
 - 2) Argyll Robertson (bilateral constricted).
 - 3) Adie's pupil (unilateral dilated/anisocoria).
 - 4) Efferent defect as in 3rd nerve palsy and sphincter muscle paralysis: Dilated (anisocoria), most common cause is atropine.
-

2. Give an account on Horner's syndrome:

Causes:

- 1) Congenital
- 2) Neck trauma or surgery
- 3) Bronchogenic carcinoma (apical lung cancer=pancoast tumour).
- 4) Brain vascular or demyelination diseases
- 5) Nasopharyngeal carcinoma.

Clinical picture:

- 1) Mild ptosis (paralysis of Muller's muscle).
 - 2) Miosis (unopposed action of sphincter due to paralysis of dilator) & anisocoria.
 - 3) Ipsilateral anhidrosis (if preganglionic lesion, before the superior cervical sympathetic ganglion due to affection of the sudomotor fibers).
 - 4) Hypochromic heterochromia (if congenital)
 - 5) Apparent or pseudo-enophthalmos.
-

3. Discuss the visual field defects of visual pathway lesion:

<i>Site of the defect</i>	<i>The defect</i>	<i>Characteristics</i>
Optic Nerve:	Optic neuritis/tumour	Central scotoma/centrocecal scotoma /monocular blindness (in optic neuritis and tumours).

	Papilledema	Enlarged blind spot/tubular field (in papilledema).
Chiasma:	Pituitary tumour	Bilateral (heteronymous) hemianopia due to destruction of crossing nasal fibers.
Optic tract:	Due to stroke (infarction-Hge) or tumour.	Crossed homonymous hemianopia (incongruous).
Optic radiation:	Temporal or parietal lobes, due to stroke or tumour	Temporal: crossed homonymous superior quadrantanopia with hemiparesis and dysphasia. Parietal: crossed homonymous inferior quadrantanopia with agnosia.
Occipital (visual) cortex:	Due to stroke (infarction-Hge) or tumour	Crossed homonymous hemianopia with macular sparing due to: - Dual blood supply (middle & posterior). - Bilateral representation of macular area. - Large area of macular representation in the cortex.

4. Discuss scotoma and tubular field:

Scotoma:

Area with decreased retinal sensitivity.

- Absolute: blind area
- Relative: decrease sensitivity to different intensities of light or certain colors.
- Negative: not seen by the patient
- Positive: seen by the patient
- + E.g above

D.D for tubular field:

- POAG
 - Advanced pigmentosa
 - Advanced papilledema
 - CRAO with patent cilioretinal arteries
-

5. Give a short account on migraine:

Definition:

Repeated attacks of headache accompanied by visual, neurological +/- GIT symptoms intervening free periods.

Risk factors:

- FH
- **OCPs**
- Certain food e.g: cheese, chocolate.
- Stress/smoking & alcoholism.

Clinical picture:

Classical migraine: 2 phases:

- **Aura:** (cerebral vasoconstriction that lasts for 10-20 min.)
 - a) Visual symptoms: disturbed vision (as zigzag lights or scotomas)/scintillations (coloured lines).
 - b) Neurological symptoms: transient speech disturbance/tingling or weakness in the limbs of one side of the body.
- **Headache:** phase (due to rebound vasodilatation):
 - a) **CCC:** unilateral-fleeting (changing sides) & throbbing.
 - b) Variable severity +/- vomiting.

Other variants:

- Ophthalmoplegic migraine
- Retinal migraine
- Migrainous neuralgia
- Headache without aura
- Aura without headache
- Hemiplegic migraine
- Status migrainosus

Treatment:

- Paracetamol, NSAIDs, ergot alkaloids.
- Sumatriptan.

MCQ:

1. Horner's syndrome:

- a. Ptosis + miosis + enophthalmos + anhydrosis
- b. Ptosis + mydriasis + enophthalmos + anhydrosis
- c. Lagophthalmos + myosis + enophthalmos + anhydrosis
- d. Diplopia + myosis + enophthalmos + anhydrosis

ANSWER : (A)

2. Clinical Picture of Horner's Syndrome doesn't include:

- a. Sympathetic paralysis.
- b. Lagophthalmos
- c. Ptosis
- d. Enophthalmos
- e. Anhydrosis

ANSWER (B)

Orbit

GIVE AN ACCOUNT ON:

1. Cavernous sinus thrombosis.

Definition:

Thrombophlebitis of cavernous sinus.

Etiology:

Infection may come from:

1. Face and orbit (ophthalmic vein).
2. Middle ear and mastoid (inferior petrosal sinus).
3. Mouth and pharynx (pterygoid plexus).
4. Blood-borne (metastasis).

Clinical picture:

a) Symptoms:

- General:

Fever, anorexia, headache & malaise (marked). **FAHM**

- Local:

1. Pain.
2. Limited ocular movement.
3. Vision:

Early good. Late decreases due to optic neuritis.

b) Signs:

1. Lid & conjunctiva: edema.
2. Proptosis:

At first unilateral, then becomes bilateral when infection spreads to the other sinus.

3. Limitation of ocular motility (paralysis of 3rd, 4th & 6th nerves in the sinus).

4. Fundus: engorged veins, papilledema or papillitis.
5. Tenderness and edema of mastoid region (to differentiate from orbital cellulitis).
6. Pupil: dilated with total ophthalmoplegia (paralysis of 3rd, 4th & 6th nerves).

N.B.

The earliest sign of affection is convergent squint (6th nerve palsy) and mastoid edema.

Treatment:

The condition is very serious and could be fatal (from meningitis, brain abscess & pulmonary infection) if not well-treated.

a) Prophylaxis:

Treatment of source of infection.

b) Curative:

1. Massive antibiotics.
2. Anticoagulants.
3. Neurosurgical approach.
4. Corneal protection from exposure keratitis.

Differential diagnosis:

	<i>Endophthalmitis</i>	<i>Panophthalmitis</i>	<i>Orbital cellulitis</i>	<i>Cavernous sinus thrombosis</i>
<i>Definition</i>	Suppurative inflammation, primarily in uveal tract (sclera is free).	Severe suppurative inflammation of uveal tract and other tissues including outer coat and soft orbital tissues.	Suppurative inflammation of orbital cellular tissue.	Thrombophlebitis of cavernous sinus.
<i>Etiology</i>	As infective iridocyclitis (see before).	As infective iridocyclitis (see before).	See before.	See before.
<i>Clinical picture:</i>				
<i>Symptoms:</i> <i>General:</i>	Headache, fever, malaise &	+ to ++	+ to ++	++++

<i>Local:</i> - <i>Pain:</i> - <i>Vision:</i>	anorexia (mild). ++ Early P.L. then decreases to no P.L. (late).	++ No P.L.	++ Good (early).	++ Good (early).
<i>Signs:</i> - <i>Lid:</i> - <i>Conjunctiva:</i> - <i>Cornea:</i> - <i>Proptosis:</i> - <i>Ocular motility:</i> - <i>Red reflex:</i>	Edema. Chemosis + ciliary injection. Hazy + kps. Absent. Normal. Yellow.	Edema. Chemosis + ciliary injection. Hazy + ring abscess. Present. Limited. Yellow.	Edema. Chemosis + injection. Clear. Present. Limited. Normal.	Edema. Chemosis + congestion. Clear. Present. Limited. Normal. Mastoid edema.
<i>Treatment:</i> <i>Antibiotics +</i>	Seeing eye: vitrectomy. Non seeing: evisceration.	Usually non seeing eye: evisceration.	Orbital abscess: drain.	Anticoagulants + neurosurgical approach.

+ Moderate.

++ Severe.

+++ Very severe.

2. Orbital cellulitis.

Definition:

Acute suppurative inflammation of orbital cellular fibro-fatty tissue.

Etiology:

1. Direct infection by:

- Penetrating wound.
- Following operation (squint/retinal detachment/..).

2. Spread of infection from:

- Sinuses.
- Teeth.
- Globe (endophthalmitis).

3. **Blood-borne:**

As in septicemia.

Causative organism:

Usually staphylococci, streptococci, pneumococci and rarely fungi.

Clinical picture:

a) **Symptoms:**

- General:

Fever, anorexia, headache & malaise.

- Local:

1. Pain.

2. Vision:

Early good. Late decreases due to optic neuritis.

b) **Signs:**

1. Edema of lid and conjunctiva:

Due to congestion in ophthalmic veins and vasodilation of ophthalmic arteries.

2. Proptosis:

Suppuration in orbit.

3. Limitation of ocular motility:

Myositis and optic neuritis lead to pain with movement.

4. An abscess maybe formed and point through:

Lower fornix or skin near lower orbital margin.

c) **Complications:**

1. Extension of infection into:

- a. Cranium (brain abscess, cavernous sinus thrombosis, meningitis, ..).

- b. Optic neuritis.

- c. Globe: endophthalmitis.

2. Central retinal vein thrombosis:

Papilledema with engorged vessels.

3. Enophthalmos with restricted eye movement:

Due to healing by fibrosis (frozen orbit).

4. Corneal exposure and ulceration due to proptosis and hypohesia.

5. General spread:

Septicemia & pyemia.

d) Differential diagnosis:

See previous table.

Treatment:

General:

Strong systemic antibiotics.

Local:

1. Hot fomentation.
2. Local antibiotics.
3. Incision and drainage if abscess is formed.
4. Treatment of corneal exposure.

3. Proptosis.

Definition:

Passive protrusion of eyeball (globe is pushed).

Active = Exophthalmos.

Etiology:

i. **Congenital:**

1. Dermoid cyst.
2. Meningo-encephalocele.

ii. **Acquired:**

1. **Traumatic:**

- a. Retrobulbar hematoma.
- b. Surgical emphysema (ruptured ethmoid bone with air escape).

- c. A-V shunt (carotid-cavernous fistula) in severe head trauma.

2. **Inflammatory:**

Acute:

- a. Orbital cellulitis.
- b. Panophtahlmitis.
- c. Cavernous sinus thrombosis.

Chronic:

- a. Specific: TB granuloma.
- b. Non specific: orbital pseudotumor.

3. **Neoplastic (benign or malignant):**

Primary (from orbital structures):

- a. Lacrimal gland tumors: adenoma, mixed tumor.
- b. Optic nerve tumors: glioma, meningioma.
- c. Hemangioma (commonest benign orbital tumor), fibroma, sarcoma, lymphoma.
- d. Extension from intraocular tumors as retinoblastoma.

Secondary from:

Breast, bronchi or prostate.

4. **Endocrinal:**

Dysthyroid ophthalmopathy (commonest cause, may start unilateral).

5. **Other causes:**

- a. Vascular: aneurysms, A-V shunts, varices (intermittent proptosis), ...
- b. Parasitic: hydatid cyst.
- c. Paralysis of extraocular muscles as 3rd nerve palsy.

Management of a case of proptosis:

i. **History:**

- 1. Trauma.

2. **Pain:**

Indicates inflammation (orbital cellulitis, panophtahlmitis or cavernous sinus

thrombosis) or malignancy.

3. Onset & course:

- a. Acute: inflammation, trauma or malignant tumor.
- b. Intermittent: varices, inflammatory pseudotumor or recurrent hemorrhage.
- c. Slowly progressive: benign tumor.

4. History of systemic disease, presence of diplopia, ...

ii. **Examination:**

1. Exclude causes of pseudoproptosis:

- a. Large globe in high myopia (esp. when unilateral).
- b. Buphthalmos.
- c. Shallow orbit in craniofacial anomalies.
- d. Contralateral enophthalmos.

2. General examination:

- ENT examination.
- Medical examination:
 - a. Enlarged lymph nodes.
 - b. Thyroid.
 - c. Look for a primary.

3. Local (ophthalmological) examination:

• **Inspection:**

a. Unilateral or bilateral:

Bilateral:

- Late cases of cavernous sinus thrombosis.
- Dysthyroid ophthalmopathy.
- Orbital reticulosis (systemic malignancy) as lymphoma and Hodgkin's disease.

Unilateral:

- Other causes.

b. Direction:

- Directly forward: optic nerve tumors.
- Forward, down and in: lacrimal gland tumor.
- Forward, down and out: frontal mucocele.
- Upward: cancer maxilla.
- c. Pulsations:
 - Ophthalmic artery aneurysm.
 - Vascular tumors (sarcoma).
 - AV shunts.
 - Meningo-encephalocele.
- d. Signs of inflammation:
 - Lid: edema.
 - Congestion, chemosis, hyperemia.
- **Palpation:**
 - a. Consistency.
 - b. Fixation of lesion.
 - c. Tenderness.
- **Ascultation:**

In carotid cavernous fistula: machinery murmur (bruit).
- **Measurement of proptosis:**
 - a. Distance between lateral orbital margin and apex of cornea is measured.
 - b. Normal = 15-18 mm. A difference of more than 2 mm is significant esp. if unilateral.
 - c. Done by simple ruler or Hertel's exophthalmometer.
 - d. Stand behind the patient and tilt the head backwards.
 - e. Notice the 2 eyes.
 - f. Which appears first is the more proptotic.
- iii. **Investigations:**
 - 1. Laboratory:
 - a. CBC e.g. for leukemia.

- b. Erythrocyte sedimentation rate (ESR).
 - c. Tuberculin test for TB. W.R. test for syphilis.
 - d. Tests for sarcoidosis.
 - e. T₃, T₄ & TSH levels.
2. Radiological:
- a. Plain X-ray:
 - Wide optic foramen: glioma of optic nerve.
 - Calcification: meningioma.
 - b. Arteriography and venography (carotid cavernous fistula).
 - c. Ultrasonography.
 - d. CT scan.
 - e. Magnetic resonance imaging (MRI).
3. Surgical:
- Biopsy (aspiration or excision in suspected tumors).

Treatment:

1. Orbitotomy and excisional biopsy:
Orbitotomy maybe:
 - a. Anterior orbitotomy for lesions in anterior half of the orbit.
 - b. Lateral orbitotomy for deeper lesions.
 - c. Trans-frontal orbitotomy for orbital apex lesions (neurosurgery).
2. Treatment of the cause.
3. Prophylactic treatment for corneal exposure.

4. Important and common causes of proptosis.

1. *Traumatic:*
 - a. Retrobulbar hematoma.
 - b. Surgical emphysema.
 - c. A-V fistula.

2. *Inflammatory:*

- a. Orbital cellulitis.
- b. Cavernous sinus thrombosis.
- c. Panophthalmitis.

3. *Endocrine:*

Dysthyroid ophthalmopathy.

4. Ocular manifestations of dysthyroid eye disease.

1. *Eyelid signs:*

- a. Dalrymple sign: lid retraction (staring look).
- b. Von Graefe's sign: lid lag (upper lid does not follow the eyeball when looking downwards).
- c. Stellwag's sign: infrequent blinking.

2. *Infiltrative ophthalmopathy:*

- a. Proliferation of orbital fat and connective tissue with retention of fluids, accumulation of mucopolysaccharides and cellular infiltration by lymphocytes and plasma cells.
- b. Enlargement of extraocular muscles due to increased mucopolysaccharides and edema with subsequent degeneration of muscle fibers, fibrosis, weakness and restricted ocular movements.

Clinical signs:

- a. Injection, hyperemia & chemosis of conjunctiva.
- b. Edema of eyelids.

3. *Proptosis:*

- a. Commonest cause of both bilateral and unilateral proptosis.
- b. The condition maybe severe leading to corneal exposure with severe ulceration and blindness.

4. *Extraocular muscle affection:*

Weakness and restricted movement in the form of defective elevation, depression, abduction and adduction.

5. *Optic neuropathy:*

- **Symptoms:**
 - a. Slowly progressive impairment of central vision.
 - b. Defective red-green color perception.

- **Signs:**

1. **Ophthalmoscopy:**

- a. Maybe normal.
- b. Disc edema and chorioretinal folds.
- c. Optic atrophy in severe cases.

2. **Visual field defects:**

Central or paracentral scotoma with or without nerve fiber layer defect (confused with primary open angle glaucoma).

3. **Afferent pupillary defect:**

Important sign of optic nerve involvement.

Management:

1. Local lubricants, dark glasses & ttt of corneal complications due to exposure.
2. Systemic steroids in early cases with painful proptosis.
3. Radiotherapy when steroids are contraindicated or ineffective.
4. **Surgical:**
 - a. Orbital decompression in severe proptosis.
 - b. Ex. ocular muscle surgery when there is diplopia in primary or reading position.

5. Differential diagnosis of acute inflammatory proptosis.

Orbital cellulitis, panophthalmitis and cavernous sinus thrombosis.

Should be differentiated from each other and from endophthalmitis. (see table).

6. Five causes of unilateral proptosis.

1. Orbital cellulitis.

2. Orbital varices.
3. Orbital pseudo-tumor.
4. Dermoid and hydatid cysts.
5. Retrobulbar hematoma.
6. Surgical emphysema.

N.B.All except:

1. Late cases of cavernous sinus thrombosis.
2. Dysthyroid ophthalmopathy.
3. Orbital reticulosis (systemic malignancy) as lymphoma and Hodgkin's disease.

7. Enophthalmos.

Definition:

Retraction of the globe into the orbit (opposite to proptosis).

Causes:

1. Senile: absorption of orbital fat.
2. Post-traumatic.
3. Post-operative.
4. Post-inflammatory.
5. Horner's syndrome (ptosis, miosis, enophthalmos and anhydrosis).

MCQ:

1. Thyroid eye disease can occur in:

- a. Hyperthyroid state.
- b. Hypothyroid state.
- c. Normal thyroid state.
- d. All of the above.

2. Proptosis is not a clinical presentation in:

- a. Panophthalmitis.
- b. Orbital cellulitis.
- c. Endophthalmitis.
- d. Grave's disease.

3. Acute proptosis may be due to:

- a. Trauma.
- b. Orbital cellulitis.
- c. Rhabdomyosarcoma.
- d. All of the above.

4. Endogenous septic focus may cause all except:

- a) Phlycten.
 - b) Hypopyon ulcer.
 - c) Iridocyclitis.
 - d) Metastatic endophthalmitis.
-

Eye injuries

ESSAY QUESTIONS:

1. Describe the ocular lesions that occur due to blunt trauma to the eye.

= A sport-man received trauma to the right eye by a tennis ball, discuss the effect of this trauma on various structures of the eye.

Cause:

Trauma by a small blunt object (fist, tennis ball, ...).

Mechanism of damage:

1. Coup & countercoup:

Coup refers to local damage at the site of impact, e.g. corneal abrasion. While countercoup refers to distant damage caused by shock rebound waves that traverse the eye to the posterior pole, e.g. commotion-retinae.

2. Antero-posterior:

Compression & horizontal expansion leads to damage, e.g. rupture globe & irido-dialysis.

Ocular lesions:

1) Orbit:

a. Traumatic proptosis:

Cause:

- Retro-bulbar hematoma.
- Peri-bulbar air (surgical emphysema due to fracture ethmoid sinus).

b. Traumatic enophthalmos:

Cause:

Fracture of the orbital floor (blow-out fracture) with escape of the orbital fat & extra-ocular muscles into the maxillary sinus.

c. Orbital cellulitis.

2) Lids:

a. Ecchymosis:

- Subcutaneous hematoma (black eye).
- Cold foment applied in 1st 24 hours (vasoconstriction) & hot foment thereafter (absorption).

b. Surgical emphysema.

c. Ptosis:

Causes:

- Mechanical: due to the weight of the lid (hematoma).
- Paralytic: due to injury of levator palpebrae superioris muscle or 3rd nerve.

d. Wounds:

- Horizontal wounds don't gape and produce small scar.
- Vertical wounds gape and need suturing.

Treatment:

Suture in layers:

- Vertical: heals by excessive scarring (cicatricial ectropion).
- Horizontal: heals by less scarring.

3) ***Conjunctiva:***

a. Wounds:

- If small (< 1 cm): leave it.
- If large: suture it.

b. Subconjunctival hemorrhage:

Maybe due to:

- Direct trauma to the eye with ruptured conjunctival vessels.
- Trauma to the head (fracture base of skull) with rupture orbit or vessels.

c. Chemosis.

	<i>Ocular trauma</i>	<i>Head trauma</i>
<i>Onset:</i>	Immediate.	Delayed.
<i>Trauma:</i>	To the eye with no proptosis.	To the head with proptosis.
<i>Conscious state:</i>	Not affected.	Lost.
<i>Site:</i>	Usually on temporal side.	Usually in fornices.

<i>Color:</i>	Bright red.	Dark red.
<i>Shape:</i>	Triangular with base towards cornea.	Triangular with apex towards cornea.
<i>Posterior limit:</i>	Seen (defined).	Not seen.

4) **Cornea:**

a. Abrasion:

- Due to damage to corneal epithelium.
- Causes severe pain and photophobia.

Treatment:

- Topical antibiotics & patching.

b. Wounds (Rupture globe with or without iris prolapse):

Less common than sclera (cornea is stronger).

Treatment:

- Suture the wound & reposit or excise the prolapsed tissues.

c. Blood staining of the cornea:

Cause:

Hyphema + rise in IOP.

Clinical picture:

- Color of the cornea is first reddish brown then greenish yellow then grey.
- The condition usually clears from the periphery to the center by phagocytic action (over 2 years or more).

Treatment:

Prophylactic:

- Control of IOP in cases of hyphema (anti-glaucoma drugs or paracentesis).
- Treatment of hyphema.

Curative:

- P.K. (penetrating keratoplasty) if blood staining becomes permanent.

d. Corneal edema:

Cause:

Endothelial & Descemet's membrane damage.

Treatment:

If mild, by hygroscopic drops (glycerin).

e. Recurrent corneal erosions:

Cause:

Erosion by corneal scratches with e.g. finger nails or paper.

Recurrence occurs with any slight trauma such as opening the eyes in the morning due to imperfect healing of basement membrane (anterior basement membrane corneal dystrophy ABMD).

5) ***Sclera:***

Laceration (rupture globe):

Site:

- Cornea: less common; as cornea is stronger than sclera.
- Limbal: weak area due to presence of canal of Schlemm.
- Sclera: commonly up & in (about 3 mm from the limbus where it's struck against trochlea).
- Trauma: commonly from down & out (less protected and more exposed).
- Eye: pushed against trochlea.

Clinical picture:

- Diminution of vision.
- Conjunctival chemosis sub conjunctival hemorrhage.
- Shallow AC.
- Hypotony.
- Abnormal site, size & shape of pupil.
- Prolapse of parts of uvea.

Complications:

- Infection (endophthalmitis).
- Intra-ocular hemorrhage.

- Prolapse of intra-ocular contents as iris.
- Sympathetic ophthalmitis.
- Lens extrusion, rupture or dislocation.
- Retinal detachment.

Treatment:

1. Immediate patching & direct pressure should be avoided.
2. Medical ttt:
 - Systemic antibiotics.
 - Anti-tetanic toxoid.
 - Anti-emetic.
3. X-ray is done to exclude presence of intraocular foreign body IOFB.
4. Surgery:
 - Hopeless cases (no P.L.): enucleation to avoid sympathetic ophthalmitis in the other eye.
 - Hopeful cases: reposit or excise the prolapsed tissue with proper suturing of the wound.

6) Iris:

a. Traumatic miosis (mild trauma):

- Transient event in all conditions.
- Caused by irritation (spasm of sphincter muscle).

b. Traumatic mydriasis (severe trauma):

- Preceded by miosis.
- Maybe transient or permanent.
- Caused by damage to motor nerves (3rd).

c. Laceration:

- At pupillary border: V-shaped (mydriatics are contraindicated as they enlarge the tear).

- Pupillary laceration of iris maybe accompanied with:

- Anti-flexion:

Torn part is rolled anterior & pigment epithelium faces the anterior chamber.

- Retro-flexion:

Torn part is rolled backward between lens equator & ciliary body.

d. Traumatic iridocyclitis.

e. Irido-donesis (tremulous iris):

Occurs with subluxation & dislocation of lens.

f. Iridodialysis or cyclo-dialysis:

Definition:

- Separation of iris root from ciliary body. This results from pressure in anterior chamber pushing iris backwards. The iris periphery (being not supported by lens) may separate.

Symptoms:

- Uniocular diplopia, except if:
 - Up (covered by lid).
 - Very small.

Signs:

- D-shaped pupil.
- Double red reflex.

Differential diagnosis:

- Malignant melanoma of the iris (normal red reflex).

Treatment:

- If no diplopia: atropine + cortisone.
- If diplopia exists:
 - Cover the defect (colored contact lens).
 - Close the defect (suture dialyzed iris to limbus).
 - Iridectomy.

g. Traumatic aniridia:

Complete avulsion of iris at its root & iris falls in bottom of anterior chamber as a black ball.

N.B. Effects on pupil are:

Traumatic mydriasis, traumatic miosis or Adie's pupil (destroyed ciliary ganglion or postganglionic parasympathetic fibers of short posterior ciliary nerves where near reflex is tonic and segmental while light reflex is absent "light-near dissociation").

7) Ciliary body:a. Hypotony:

Due to ciliary body shock or cyclo-dialysis "separated from the scleral spur" (suppression of aqueous formation).

b. Glaucoma, due to CB laceration:

- Hyphema.
- Healing by fibrosis closing the angle (angle-recession glaucoma).

c. Spasm of accommodation:

With temporary myopia.

d. Paralysis of accommodation:

With blurred near vision.

e. Intra-ocular hemorrhage.**8) Choroids:**a. Choroidal effusion or hemorrhage.b. Choroidal rupture:

- Site: temporal to disc.
- Shape: crescentic with its concavity towards the disc.
- Color: early, edges are covered with hemorrhage. Late, white (showing sclera).
- Crossed by: retinal vessels (retina is intact).
- Fate: good, if away from fovea.
- TTT: rest + vitamin C.

c. Traumatic choroiditis.

- d. Choroidal detachment from hypotony.

9) *Lens:*

- a. Subluxation: partial rupture of zonules.
b. Dislocation: total rupture of zonules.

Signs & symptoms of both:

- Decreased V.A.
- Uniocular diplopia in subluxation.
- High degree of astigmatism.
- Impaired accommodation.
- Tremulous iris (loss of lens support).

Complications:

- Pupil-block glaucoma (glaucoma inversus) with anterior lens dislocation.
- Phaco-anaphylactic glaucoma with posterior dislocation.

Management:

- Dislocated lenses are removed to avoid complications.

- c. Traumatic cataract:

- Vossius ring (impression of pupillary border of iris with its pigment on anterior lens capsule).
- Anterior & posterior cortical opacities (posterior are more common due to thinner capsule).
- Rosette-shaped cataract (pathognomonic of blunt trauma, caused by disruption of lens architecture at cortical sutures).

10) *Anterior chamber:*

- a. Anterior dislocation.
b. Hyphema:

Definition:

- Blood in anterior chamber due to ruptured iris vessels

Source:

- Lacerated iris & ciliary body.

Complications:

- 2ry glaucoma.
- Blood staining of cornea.

TTT:

1. Rest in bed (semi-sitting position).
2. Bandage of both eyes.
3. Daily monitoring of IOP.
4. Medical ttt:
 - a. Local: steroids (iritis)& beta blockers (control IOP).
 - b. General: oral aminocaproic acid (anti-fibrinolytic, 50-100 mg/kg every 4 hours) + vitamin C & chemotrypsin.
 - c. NSAIDs are avoided.
5. Surgery (for 2ry glaucoma):
 - a. Paracentesis.
 - b. Evacuation of hyphema (washing by irrigation/aspiration).

N.B.

- No mydriatics; as they lead to:
 - Decreased absorptive surface of iris.
 - Angle closure glaucoma.
- No miotics:
 - Pupil block glaucoma.
 - Synechia.
- Both lead to iris movement with loosening of the clot resulting in re-bleeding.

11) Vitreous body:

- a. Vitreous hemorrhage.
- b. Vitreous opacification (Musca volitans) due to:
 - Hemorrhage.
 - Coagulated protein.

- c. Vitreous loss through rupture globe with tractional retinal detachment.
- d. Avulsion of vitreous base causing retinal dis-insertion.

12) Retina:

- a. Retinal tears (dialysis or giant tear): leading to retinal detachment.
- b. Retinal hemorrhage: intra-retinal or subhyaloid.
- c. Retinal edema: commotion-retinae = Berlin's edema (discussed later).
- d. Retinal detachment maybe:
 - Rhegmatogenous: due to retinal tear.
 - Exudative: due to severe hypotony.
 - Tractional: due to vitreous loss & incarceration in scleral wound.

13) Optic nerve:

- a. Avulsion of optic nerve with twisting injuries.
- b. Optic nerve sheath hemorrhage.
- c. Edema of optic nerve with hypotony.
- d. Optic atrophy (usually primary).

14) IOP:

- a. Traumatic glaucoma (cause).
- b. Traumatic hypotony (CB shutdown).

15) Extra-ocular muscles:

Squint.

N.B.

- Anterior segment lesions = cornea, iris, ciliary body and lens (structures in front of vitreous).
- Posterior segment lesions (behind the lens) = vitreous, retina, choroid & optic nerve.

2. Commotio-retinae (Berlin's edema):

Definition:

Retinal edema following ocular blunt trauma.

Cause:

A counter-coup to posterior pole of the eye leading to compression of posterior pole vessels that results in retinal edema (most marked in central part).

*Clinical picture:**Symptoms:*

Rapid (acute) drop of vision (up to hand motion HM).

Signs:

1. Sluggish pupil.
2. Fundus examination: cherry red spot +/- scattered retinal hemorrhages.
 - Greyish-white color (due to edema collected mainly in ganglion cell layer).
 - In fovea (no ganglion cells), so still shows color of the choroid.

Fate:

1. Spontaneous resolution within month.
2. Macular degeneration or hole with severe loss of vision in some cases.

Differential diagnosis:

1. From other causes of cherry red spot:
 - a. CRAO.
 - b. Quinine poisoning,
 - c. Tay Sachs disease (muco-lipidosis).
2. From other causes of rapid diminution of vision (esp. within hours) as:
 - a. Acute congestive glaucoma (painful).
 - b. Vitreous hemorrhage (painless).
 - c. CRVO (painless).

Treatment:

1. Rest.
2. If severe, give systemic steroids.

3. Blow-out fracture of orbital floor.

Signs & symptoms:

1. Peri-bulbar air (surgical emphysema due to fracture ethmoid sinus).
2. Traumatic enophthalmos with herniation of fat into maxillary sinus.
3. Diplopia & defective eye movement (esp. elevation) d.t. entrapment of an extraocular muscle in the fractured site.

Management:

1. CT of the orbit.
2. Immediate surgery is rarely required.
3. Oral antibiotics (to protect against sinus bacteria & development of orbital cellulitis).
4. Surgery will aim at correcting persistent problems as diplopia and disfiguring enophthalmos.

4. Enumerate causes of decreased visual acuity following ocular blunt trauma (2000).

1. Sudden loss of vision:

Rupture globe.

2. Rapid diminution of vision:

Hours:

- a. Commotio-retinae.
- b. Vitreous hemorrhage.

Days:

- a. Retinal detachment.
- b. Choroiditis.
- c. Iridocyclitis.

3. Gradual diminution of vision:

Primary optic atrophy.

5. Sympathetic ophthalmitis.

Definition:

Bilateral inflammation of uveal tract following perforating trauma to one eye, in which part of uveal tract is involved leading to marked diminution of vision.

- Traumatized eye is called: exciting eye.
- Other eye is called: sympathizing eye.

Etiology:

1. *Predisposing factors:*

Incidence is increased with:

- a. Injury to ciliary body (dangerous zone) with uveal prolapse.
- b. Retained intraocular foreign body IOFB.
- c. Incarcerated uveal tissue in perforating wound.

2. *Theories:*

a. Allergic theory:

- The uveal tissue is normally isolated from immune system of the body.
- An ocular injury will liberate the uveal pigment (antigen) to the circulation.
- Antibodies formed against uveal pigment will attack both eyes.
- More accepted nowadays, since:
 - i. Latent period is 4-8 weeks (average time needed by immune system to form antibodies).
 - ii. Patients have cutaneous hypersensitivity to uveal pigments.
 - iii. Disease is extremely rare in ocular suppurations (as endophthalmitis) where uveal pigment is destroyed by pus.

b. Infective theory:

- The organism (maybe virus or bacteria) reach the exciting eye with trauma & reaches the other eye via optic nerve $\Rightarrow$ chiasma $\Rightarrow$ other optic nerve.
- Not accepted nowadays (no organism is isolated from lesions).

Clinical picture:

Onset:

4-8 weeks after trauma (maybe up to years).

Prodromal picture:

Sympathetic irritation, better seen in sympathizing eye.

Symptoms:

1. History of trauma to one eye in most patients.
2. Pain, lacrimation, photophobia & defective vision.
3. Loss of accommodation (indistinct near vision).
4. Symptoms are bilateral, but start in exciting eye followed (days-weeks) by sympathizing eye.

Signs:

1. Signs of trauma in exciting eye (as retained FB).
2. Signs of bilateral iridocyclitis (keratic precipitates) with variable degree of severity.

Full picture:

1. The condition progresses into:
2. Severe bilateral pan-uveitis.
3. Secondary glaucoma, complicated cataract & retinal detachment.
4. Finally, atrophie bulbi.

*Treatment:**1. Prophylactic:*

- a. If injured eye is hopeless: enucleation.
- b. If injured eye is hopeful:
 - Excise prolapsed tissues.
 - Remove any IOFB.
 - Proper suturing of wound.
 - Follow up.

2. Curative (if prodromal symptoms & signs appear in normal eye):

- a. Topical atropine & cortisone for both eyes.
- b. Enucleate traumatized eye if medical ttt failed within 2-3 weeks (help to ameliorate the condition and save the sympathizing eye).

N.B.

Sympathetic ophthalmitis never occurs if there is suppurative choroiditis (endo or panophthalmitis) as suppuration will destroy the uveal pigment (antigen).

6. Ocular effect of Intraocular Foreign bodies.

1) Mechanical effects, depend on:

- Route of entry (may affect cornea, sclera, uvea, retina or optic nerve).
- Size (large FB $\Rightarrow$ more damage)
- Shape (ragged FB $\Rightarrow$ more damage)
- Velocity.

2) Infection:

- Not common (many IOFB "esp. metallic" are sterile due to heat generated during their emission).
- Sterile inflammation is common especially with pure copper.

3) Chemical effects:

- If chemically inert (e.g.: glass) $\Rightarrow$ it'll be surrounded by fibrosis.
- If chemically active:
 - Iron $\Rightarrow$ siderosis.
 - Copper $\Rightarrow$ chalcosis.

4) Sympathetic Ophthalmitis (discussed before).

(مواد ثانوية عامة) فيزياء (mechanical) - أحياء (infection) - كيمياء (chemical) - علم النفس (sympathetic).

7. Describe effect of chemical injuries on the eye & management.

Clinical picture:

- Pain, photophobia, lacrimation & diminution of vision.

Mild to moderate exposure:

- Lid edema.
- Chemosis.
- Conjunctival injection.
- Corneal abrasions.
- Anterior uveitis.

Severe exposure:

- Conjunctival & episcleral whitening (coagulative necrosis).
- Corneal edema & opacification with corneo-scleral melting.
- Severe iritis.
- Secondary glaucoma.
- Posterior segment destruction.

Effects & complications:

1. Ulceration: Skin, conjunctiva & cornea.
2. Scarring:
 - a. Lid: cicatricial entropion or ectropion.
 - b. Conjunctiva: symblepharon & xerosis.
 - c. Cornea: opacities & xerosis.
3. Secondary Glaucoma due to:
 - a. Iritis (in alkali burn).
 - b. Conjunctival fibrosis: including aqueous veins.
4. Atrophy bulbi (loss of vision): this depends on:
 - a. Concentration.
 - b. Duration: interval between injury & 1st aid application.
 - c. Penetration:
 - Acids are less serious than alkali because they coagulate & precipitate proteins; thus act as a barrier that prevents further penetration (superficial burns) "oral question".
 - Alkalis have rapid penetration (less than 1 minute) & combine with cell membrane lipids leading to cell disruption & tissue necrosis.

*Management:**1. 1st Aid:*

If Chemical nature of substance is known: Wash with antidote:

- Strong acids $\Rightarrow$ NaHCO_3 3%.
- Strong Alkali $\Rightarrow$ Boric acid lotion 4%.

- Iodine $\Rightarrow$ starch solution or milk.
- Lime $\Rightarrow$ Pick particle with forceps, then wash with
 - o EDTA (0.1%).
 - o Conc. sucrose solution (neutral lime saccharate).
 - o **Never with water.**
- Aniline $\Rightarrow$ 10% alcohol then 10% glycerin.

If Chemical nature is unknown: Wash with either water or milk:

- Tap water $\Rightarrow$ dilute chemical substance.
- Milk $\Rightarrow$ dilution + buffer acids, alkalis & Iodine.
- It's better to place an eye speculum & topical anesthesia before irrigation. Lower lid is pulled down & upper lid is everted to irrigate the fornices.
- Conjunctival pH should be tested 10 minutes after cessation of irrigation using litmus paper. Irrigation should be continued till reaching neutral pH (7.0).

2. *Drugs (topical & general):*

- Atropine for corneal ulceration.
- Antibiotic to prevent 2ry infection.
- Oral acetazolamide or topical beta blockers to treat any rise in IOP.
- Vitamins A & C.
- Avoid steroids in case of corneal abrasions.

3. *Prevention of symblepharon with glass rods.*

4. *TTT of complications (e.g. tarsorrhaphy for lagophthalmos in case of severe damage of lids).*

5. *If impending perforation, do therapeutic keratoplasty.*

8. **Effects of metallic foreign body penetrating the eye.**

Metallic foreign body = iron, copper or lead.

Extraocular effects:

Commonest sites:

Cornea, fornix & sulcus subtarsalis.

Complications:

- Corneal ulcers.
- Corneal opacities (if Bowman's membrane is damaged).

Treatment:Removal (under surface anesthesia):

- Use a glass-rod with its tip wrapped with a piece of cotton & sweep the corneal surface.
- If embedded, use a needle or foreign body spud).

*Intraocular effects:**1. Mechanical effects, depend on:*

- a. Route of entry (may affect cornea, sclera, uvea, ...).
- b. Size (large FB $\Rightarrow$ more damage)
- c. Shape (ragged FB $\Rightarrow$ more damage)
- d. Velocity.

2. Infection:

- a. Not common (sterile due to heat generated during their emission).
- b. Sterile inflammation is common especially with pure copper.

3. Chemical effects:

Metallic foreign bodies are chemically active:

- Iron $\Rightarrow$ siderosis.
- Copper $\Rightarrow$ chalcosis.

Both are discussed below.

*4. Sympathetic Ophthalmitis (discussed before).***9. Siderosis bulbi.***Definition:*

Toxic effect of iron on the eye.

Etiology:

1. Iron IOFB.

2. Intra-ocular hemorrhage.

Mechanism & clinical picture:

1. Iron is oxidized (rust $\Rightarrow$ ferrous & ferric oxide).
2. Rust separates from the foreign body, dissolve in tissue fluids & circulate inside the eye leading to:
 1. Staining of the tissues with rusty (brown-red) color.
 2. Entering the cells (esp. epithelial cells) producing toxic effects on cellular proteins & inactivation of intracellular oxidative enzymes, leading to:
 - Symptoms of diminution of vision.
 - Night blindness.
 3. Deposition in the angle & scarring of trabecular meshwork causing secondary glaucoma.

Signs:

1. Cornea:
Krukenberg spindle (iron in endothelium).
2. Iris & ciliary body:
Atrophic changes (mydriasis) & heterochromia (ipsilateral iris is darker).
3. Lens:
Siderotic cataract (not true cataract, caused by iron in subcapsular epithelium).
4. Retina (rods):
Pseudo-retinitis pigmentosa (night blindness).
5. Optic nerve:
Consecutive optic atrophy leading to blindness.
6. Secondary glaucoma.

Treatment:

1. Early removal of the foreign body.
2. Treatment of complications, e.g. cataract & glaucoma.
3. If eye is blind & painful, enucleate to avoid sympathetic ophthalmitis.

10. Chalcosis bulbi.

Definition:

Toxic effect of copper in the eye.

Mechanism:

1. Pure copper produces severe (sterile) inflammation that stimulates endophthalmitis.
2. Copper is oxidized into CuO which separates from the FB, dissolve in tissue fluids & circulate inside the eye, leading to staining of ocular tissues (esp. collagen & basement membranes) with yellow-green color.
 - a. Lens: sun-flower cataract.
 - b. Cornea: Keyser-Fleischer ring (golden ring at corneal periphery dt. deposition of copper in Descemet's membrane).

Treatment:

As siderosis bulbi.

MCQ:

1. The first line of management of an acidic chemical injury is:

- a. Wash with water immediately.
 - b. Do not touch the eye, refer to a specialist.
 - c. Give IV steroids then refer.
 - d. Look for an alkaline antidote to wash with.
-

2. Hyphema is:

- a. Blood in vitreous cavity.
- b. Pus in anterior chamber.
- c. Blood in anterior chamber.

- d. Lens dislocation.
-

3. Iridodialysis:

- a. Separation of iris root from ciliary body.
 - b. Patient complains from binocular diplopia.
 - c. Pupil is round and regular.
 - d. Always associated with retinal dialysis.
-

4. Scleral rupture is usually up and nasal due to:

- a. Trauma usually comes from up and nasal.
 - b. Eye ball is pushed against trochlea.
 - c. This is the strongest part of sclera.
 - d. Superior rectus is attached at this site.
-

5. Rupture of choroid affects the vision markedly:

- a. In most cases.
 - b. If is underlying fovea.
 - c. If caused by blunt trauma.
 - d. If covered with hemorrhage.
-

6. Patient received a tennis ball hit to his eye which used to have 6/6 vision. External eye examination showed no abnormalities. Vision is H.M. and red reflex is normal. Possible diagnosis is:

- a. Commotio-retinae.
- b. Traumatic cataract.
- c. Vitreous hemorrhage.
- d. Subconjunctival hemorrhage.

Errors of refraction

DISCUSS

1. Definition, etiology, types, symptoms, signs, fundus changes and treatment of myopia

Definition

Error of refraction in the eye in which with accommodation is completely relaxed; parallel rays come to focus in front of the retina.

Rays coming out of the retina leaves the eye convergent and meet in front of the eye.

The point where they meet is called the far point of the eye and its distance of the eye depends on the degree of myopia. The higher the myopia the shorter would be this distance.

Far point and the retina are conjugate foci.

Etiology

1- Axial

- eye is longer than average

2- Refractive:

- a- **curvature:** due to increased curvature of cornea or lens
- b- **Index:** due to increased refractive power of the lens, as in cases of DM and cataract
- c- **Anterior displacement of the lens**

Types

1- Simple

- Physiological variant of normal
- usually doesn't progress after adolescence when it reached 5 or 6 diopters

2- Progressive

- Increases steadily up to 25 years or beyond and may reach more than 25 diopters
- Hereditary and more common in women, and has a racial tendency

3- Congenital

- Child is born with an abnormally long eye giving a refraction of about -10 diopters but progression is rare

Symptoms

- 1- Indistinct far vision
- 2- Screwing of eyelids to simulate a pinhole that increased depth of focus
- 3- Muscular asthenopia
- 4- Symptoms due to degenerative changes as:
 - muscavolitans
 - photopsia
 - poor night vision

Signs

- 1- **Visual acuity:**
 - decreased far vision
- 2- **Retinoscopy:**
 - to locate far point of vision
- **In progressive (degenerative) myopia:**
 - 1- Large eye (pseudoproptosis)
 - 2- Apparent convergent squint
 - 3- Exophoria and exotropia

3- Fundus changes

- a- **Vitreous:**
 - Vitreous liquefaction and opacities (floaters)
- b- **Retina and choroid:**
 - Tigroid fundus due to decreased retinal pigmentation
 - Temporal crescent due to separation of the retina and choroid from the temporal margin of the disk
 - Lacquer cracks due to breaks in Bruch's membrane
 - Macular hemorrhage
 - Chorioretinal scars and atrophic patches

- Fuch's spot: a dark foveal spot causing sudden drop of vision
- Peripheral chorioretinal degenerations, retinal breaks and retinal detachment

Treatment

- **Eyeglasses:**
 - Concave spherical (minus) lenses: to move the image back to the retina
 - Children should wear their spectacles constantly for their normal development
Otherwise, they would tend to become introspective with more interest in near work
- **Contact lenses** can be used if desired by the patient
- **Refractive surgery** can be done when indicated

Complications of myopia

Occur only in high axial myopia. They include:

1- Squint:

- Pseudo-convergent squint
- True divergent squint (exophoria – exotropia)

2- Complicated cataract:

- Posterior subcapsular
- Nuclear

3- Vitreous complications:

- Vitreous liquefaction and opacities (floaters)

4- Retinal and choroidal complications:

- Tigroid fundus due to decreased retinal pigmentation
- Temporal crescent due to separation of the retina and choroid from the temporal margin of the disk
- Lacquer cracks due to breaks in Bruch's membrane
- Macular hemorrhage
- Chorioretinal scars and atrophic patches
- Fuch's spot: a dark foveal spot causing sudden drop of vision
- Peripheral chorioretinal degenerations, retinal breaks and retinal detachment

5- Scleral complications:

- Posterior staphyloma: high myopia is the only cause of posterior staphyloma

2. Fundus changes in high myopia

See above

3. Hypermetropia (definition, etiology, symptoms, signs, complication, component and treatment)*Definition*

Error of refraction in the eye in which with accommodation is completely relaxed; parallel rays come to focus behind the retina.

Rays coming out of the retina leaves the eye divergent and can meet only in a virtual point behind the eye.

The point where they meet is called the far point of the eye and its distance of the eye depends on the degree of hyperopia.

Far point and the retina are conjugate foci.

*Etiology***1) Axial**

- eye is shorter than average

2) Refractive:

a- curvature: due to decreased curvature of cornea or lens

b- Index: due to decreased refractive power of the lens, as in cases of DM and with aging

c- Posterior displacement of the lens

d- Aphakia

*Components***1- Total hyperopia (+8D)**

Amount of hyperopia under the effect of atropine (cycloplegic) i.e. with the eye tone of ciliary muscle is completely lost

2- Latent (+1D)

Amount of hyperopia corrected by normal tone of ciliary muscle

3- Manifest

All amount of hyperopia not corrected by the normal tone of the ciliary muscle (7D)

= total-latent

Corresponds to the highest plus lens giving the maximum visual acuity.

It can be divided into:

a) Facultative +3D

➔ Can be overcome by accommodation

b) Absolute 4D

➔ Cannot be overcome by accommodation.

Corresponds to the lowest plus lens giving the maximum visual acuity.

Symptoms

- 1- Blurring (near > far)
- 2- Accommodatable asthenopia (eye strain)
- 3- Early onset of presbyopia

Signs

- 1- Retinoscopy:
 - locates the far point
- 2- Slit lamp examinations:
 - shows small globe and shallow anterior chamber
- 3- Fundus examination:
 - Water silk reflex
 - Tortuous non-leaky blood vessels
 - Pseudoneuritis

Complications:

- 1) Squint:
 - Pseudo-divergent
 - High degree apparent divergent squint and manifest convergent squint (esophoria and esotropia)

- 2) Angle closure glaucoma due to shallow anterior chamber and narrow angle

Treatment

- Eyeglasses:
 - Convex spherical (plus) lenses: to move the image forward to the retina
- Contact lenses can be used if desired by the patient but usually are less tolerated than myopia
- Refractive surgery can be done when indicated but the procedure is less successful than myopia

4. Astigmatism

Definition

Error of refraction in the eye in which with accommodation is completely relaxed; parallel rays do not form a point focus on the retina. Instead, eye produces an image with multiple focal points or lines i.e. eye has different refractive powers in different meridians

Types:

1) Regular

- 2 major meridians are perpendicular to each other; one with the greatest refractive power and the other with the least refractive power
- There's gradual change of the power from the least to the greatest meridians
- Usually the vertical meridian is steeper (greatest refractive power), and the condition is called Astigmatism With the Rule
- When the vertical meridian is flatter (less refractive power) the condition is called Astigmatism Against the Rule.

So, we have

- 1- Simple (myopic or hyperopic) astigmatism:
 - If only one meridian is ametropic
- 2- Compound (myopic or hyperopic) astigmatism:
 - If both principal meridians are of the same ametropia
- 3- Mixed (myopic or hyperopic) astigmatism:
 - If one meridian is myopic and the other is hyperopic

Etiology:

Due to irregularities in curvature of cornea or lens

- Corneal astigmatism (most of cases):
 - It's mostly naturally occurring as a physiological variant
 - May be induced by surgical or traumatic scars
 - It may also occur due to ectatic diseases of the cornea as early keratoconus
- Lenticular astigmatism: subluxation of crystalline lens or tilted IOL
- Posterior staphyloma may induce astigmatism

Clinical picture:

a) Symptoms:

- Accommodative asthenopia due to subsequent contraction relaxation of ciliary muscle
- Indistinct vision
- Head tilt
- Distorted object (difference in curvature so circle appears oval)

b) Signs:

- Patient reads some types of visual acuity charts and cannot read other types on the same line
- Some opening of ring in the same line is not seen (in Landolt's chart)
- Astigmatic fan, some lines appear sharp black, others are blurred and grey
- Placido disc: shows irregular circles in cases of irregular astigmatism
- Keratometer: the curvature (power) can be measured
- Ophthalmoscope (the optic disc appears oval)
- Retinoscopy:
 - by which we can measure power of each meridian
 - point of neutralization is different in different meridians
- Autorefractometer
- Gross signs e.g. keratoconus, corneal opacities, subluxation

Treatment of regular astigmatism by:

- 1- Glasses

- cylindrical with its axis perpendicular to meridian to be corrected for simple astigmatism
- sphero-cylindrical for compound and mixed astigmatism
- 2- Special soft contact lens can be used but with poor tolerability
- 3- Refractive surgery is effective in limited cases

2) Irregular:

- When the power is different in different meridians without any rule e.g., scars and ectasia
- Meridians of highest and least power are not perpendicular to each other
- Transmission from the highest to the least is not gradual

Etiology:

1. Corneal (irregular):
 - Scar: nebula
 - Ectasia: Keratoconus & Keratectasia.
2. Lens:
 - Incipient cataract
 - Severe subluxation
 - Severe tilting in IOP
3. Retina: posterior staphyloma

Clinical picture:

a) Symptoms:

Marked blurring and distortion.

b) Signs:

1. **Slit lamp examination: of the cause**
2. Retinoscopy (red reflex appears as):
 - Scissoring in nebula
 - Spinning or rotatory in keratoconus
3. Keratometry: distorted mires.
4. Placido disc: irregular mires.

5. Corneal topography.

Treatment of Irregular astigmatism:

- 1- Rigid contact lens: to convert the irregular anterior surface into a regular one
- 2- Penetrating keratoplast: can be done if vision does not improve with rigid contact lens

5. Definition, clinical picture, and correction of anisometropia

Definition

Condition of refraction in which difference in refractive power in both eyes more than 4 diopters

Clinical picture:

- 1- Asthenopia
- 2- Difficulty in binocular vision
- 3- Squint (divergence)

Treatment

- 1- Glasses can be used after under correcting the eye with a higher error at the expense of good vision
- 2- Contact lens reduce the difference in retinal image to about 6%
- 3- IOL produce difference of less than 1% and refractive surgery makes the difference negligible

6. Anisokonia

It's difference on size of the retinal images.

It occurs if refractive correction of anisometropia is attempted by glasses because they produce a difference in the retinal images by about 25%.

This is rarely tolerable when the difference is more than 4 diopters

7. Asthenopia

Definition

Asthenopia or eyestrain is a group of symptoms noticed with visual tasks chiefly after close

work, especially in the evening by artificial illumination

The symptoms include:

- 1- Eye ache and burn
- 2- Dry sensation leading to frequent blinking
- 3- Lacrimation
- 4- Hyperemia of the conjunctiva and lid margin
- 5- Headache, usually frontal

Causes

- a- Accommodative asthenopia:
 - Hypermetropia
 - Astigmatism
 - Presbyopia
 - Anisometropia
- b- Muscular asthenopia:
 - due to disproportion between convergence and accommodation in heterophoria

8. Presbyopia

Definition

Recession of the near point due to loss of accommodation with aging making near vision uncomfortable

Mechanism

Crystalline lens becomes hard with age and loses its ability to change its dioptric power

Symptoms

- 1- Difficult near vision:
 - Usually occurs around the age of 45 in a previously emmetropic eye.
 - A previously hypermetropic patient may need a presbyopic correction at an earlier age.
 - A previously myopic patient may need a presbyopic correction at older age.
 - A myope of -3 would not need a presbyopic correction

2- Accommodative asthenopia

Treatment

1- Eyeglasses:

- Plus lenses added to the are correction: are used to compensate for the lost automatic focusing power of the crystalline lens

a- The near correction may occupy the whole spectacle frame, to be used only for reading

b- The near correction may occupy only the lower half of a Bifocal spectacle, to be used for both near and far vision (upper part for far, lower part for near)

c- A multifocal spectacle contains progressive lenses that correct for all distances by gradual change of power from furthest to nearest towards the lower segment of the lens

2- Multifocal contact lenses:

- may have been tried with limited success

3- Multifocal intraocular lenses:

- may be used during cataract extraction

9. Amblyopia.

Definition:

Unilateral or rarely bilateral decrease in best corrected visual acuity in absence of eye pathology or visual pathway disease.

= Impaired vision in absence of any organic disease, most likely due to lack of continuous use of one or both foveas for vision.

Etiology & types:

Main cause is either:

1. Vision deprivation.
2. or Abnormal binocular interaction.

A. Stimulus deprivation amblyopia:

Caused by media opacity as in unilateral or bilateral asymmetrical cataract, or unilateral severe ptosis covering pupil.

B. Anisometropic amblyopia:

Caused by marked difference in refractive error between both eyes leading to abnormal binocular interaction (patient ignores blurred image of one eye).

C. Strabismic amblyopia:

Strabismus (squint) leads to abnormal binocular interaction & occurrence of diplopia leading to suppression of the image of squinting eye to avoid this diplopia.

Diagnosis:

- Visual acuity is less in the amblyopic eye by 2 lines or more on Landolt's chart, in absence of organic cause.
- Neutral density filter:
Causes significant drop by 2 lines or more in case of organic lesion (but no significant drop in amblyopia).

Treatment:

Aim:

Forcing patient to depend on amblyopic eye for vision.

Critical period during which amblyopia can be reversed is up to:

- 7-8 years in strabismic amblyopia.
- 11 years in anisometropic amblyopia.

By:

1. Occlusion therapy (covering normal eye). One week per each year of life, then monitor visual acuity in both eyes to avoid inducing amblyopia in normal eye.
2. Pinnalization in uncooperative child: atropine eye drops in sound eye so that the child won't see well with it).

10. Discuss alcohol toxic amblyopia

Etiology:

It's due to ingestion of methyl alcohol or inhalation of its fumes.

Pathogenesis:

Breakdown of methyl alcohol in the body to formaldehyde and formic acid leading to acidosis and anoxia and finally degeneration of the ganglion cells and nerve fibers.

Clinical picture:

- Acute stage: nausea, vomiting, headache, giddiness, coma and death.
- If the patient survives###, vision is usually lost due to optic atrophy.

Treatment of toxic optic neuropathy:

1. Stop exposure.
2. Acute stage of methyl alcohol toxicity: Gastric lavage with ethyl alcohol.
3. Combating acidosis by sodium bicarbonate local and IV may be life and vision saving.
4. Vasodilators and vit B complex.

MCQ:

1- Astigmatism can be diagnosed with

- a) Placcido disc
- b) Corneal topography
- c) Astigmatic fan
- d) All of the above

2- Best option for management of anisometropia in unilateral Aphakia is

- a) Glasses
 - b) IOL implantation
 - c) Contact lenses
 - d) Radial keratotomy
-

Squint (Strabismus)

ESSAY QUESTIONS:

1. Mention origin, insertion, nerve supply and actions of extraocular muscles.

- *Extraocular muscles are:*

Four recti: superior, inferior, medial & lateral rectus.

Two obliques: superior and inferior oblique.

- *Origin:*

The four recti and superior oblique:

Arise from a common tendinous ring called (annulus of Zinn) around the optic foramen.

Inferior oblique:

Arises from floor of the orbit lateral to the opening of nasolacrimal duct.

- *Insertion:*

Four recti:

Pass forwards to be inserted in sclera in front of the equator at variable distances from the limbus.

Superior oblique:

Passes forwards and medially to reach the trochlea at upper medial part of the orbit.

Then bends backwards, downwards and laterally to be inserted in upper posterolateral part of the eye.

Inferior oblique:

Passes backwards and laterally below the globe to be inserted into lower posterolateral part of the eye, 5 mm from optic nerve, almost over the macula.

- *Action:*

Medial rectus:

Adduction.

Lateral rectus:

Abduction.

Superior rectus:

Elevation (primary action).

Adduction and intorsion (subsidiary action).

Inferior rectus:

Depression (primary action).

Adduction and extorsion (subsidiary action).

Superior oblique:

Intorsion (primary action).

Depression and abduction (subsidiary action).

Inferior oblique:

Extorsion (primary action).

Elevation and abduction (subsidiary action).

NB.

- Superior muscles are intorters. Inferior muscles are extorters.
- Recti are adductors. Obliques are abductors.
- On abduction: recti are only elevators and depressors.
- On adduction: obliques are only elevators and depressors.
- *Nerve supply:*
 - Oculomotor (3rd) nerve supplies medial, superior, inferior rectus & inferior oblique.
 - Trochlear (4th) nerve supplies superior oblique (SO4).
 - Abducent (6th) nerve supplies lateral rectus (LR6).
- *Blood supply:*
 - Muscular branches of ophthalmic artery.

2. Definition & types of squint.

Definition:

A condition in which the visual axes of the two eyes are not directed to the same object.

Visual axis: the line connecting fovea & object of fixation, passing through nodal point (nasal to optic axis).

Nodal point: The point at which rays entering the eye undergoes no refraction, just near to

posterior pole of lens.

Types:

1. Apparent squint.
2. True squint:
 - a. Latent squint (-phoria).
 - b. Manifest squint (-tropia): paralytic or concomitant.

NB.

Optic axis: the line connecting center of cornea, nodal point & center of retina (posterior pole).

Angle alpha: angle between optic & visual axis.

Emmetrope: +5

Hypermetrope: > +5 (apparent divergent squint).

Myope: < +5 or even negative (apparent convergent squint).

3. Apparent squint (Pseudo-squint).

Definition:

A condition in which the person appears to have deviated eye (false impression), although the 2 visual axes of both eyes are directed to same object = orthophoric.

Etiology:

1. Apparent convergent squint:
 - a. Epicanthus: a skin fold over the medial canthus (congenital or racial).
 - b. High myopia (negative angle alpha).
 - c. Small interpupillary distance (normal IPD = 55-70 mm).
2. Apparent divergent squint:
 - a. Latera; ankyloblepharon: adhesion between lid margins.
 - b. High hypermetropia: large positive angle alpha.
 - c. Large IPD (hypertolerism = wide distance between the two eyes).
3. Apparent upward deviation:

Ptosis.

4. Apparent downward deviation:

Lid retraction.

Diagnosis:

1. Presence of a cause.
2. Cover test: no movement.
3. Corneal light reflex: normal (centered).

Treatment:

Treatment of cause only (no squint).

3. Heterophoria (Latent squint).

Definition:

A condition in which the eye has a tendency to deviate due to EOM Imbalance, but this tendency is checked (prevented) by the brain subconsciously to maintain binocular vision. When the patient is fatigued and the brain loses interest in binocular vision latent squint becomes manifest.

Etiology:

EOM imbalance:

1. Uncorrected errors of refraction:

Leading to disproportion between accommodation and convergence.

- Hypermetropia:
 - a. Excessive accommodation to see clearly.
 - b. Increased convergence.
 - c. Latent convergent squint.
- Myopia:
 - a. Relaxed accommodation.
 - b. Lack of convergence.
 - c. Latent divergent squint.

2. Mild weakness of one or more EOM:

But not sufficient to produce manifest squint.

Types:

1. Exophoria:

The eye tends to deviate outwards.

2. Esophoria:

The eye tends to deviate inwards.

3. Hyperphoria:

The eye tends to deviate upwards.

4. Hypophoria:

The eye tends to deviate downwards.

5. Excyclophoria:

The eye tends to roll out.

6. Incyclophoria:

The eye tends to roll in.

*Clinical picture:**Symptoms:*

- Compensated cases: show no symptoms (most cases).
- Decompensated cases may show:
 1. Symptoms due to trial to maintain binocular vision (muscular athenopia or eye strain) after prolonged close work. The eyes are red with ocular pain, headache, lacrimation, recurrent sty and blepharitis.
 2. Symptoms due to failure to maintain binocular vision (latent $\Rightarrow$ manifest) in case of lost interest for fixation or fatigue:
 - a. Intermittent diplopia.
 - b. Running letters due to intermittent diplopia.

*Diagnosis:*Principle:

Depends on abolishing the binocular vision by covering one eye, presenting different images to each eye, so the brain will lose interest of binocular vision and latent squint will become manifest squint.

1. Cover-uncover test:

- o Ask the patient to fix an object (torch).
- o Cover then uncover one eye.
- o If this eye deviates under the cover and correct its position with uncovering so it's latent squint.

2. Maddox red (for far):

- o Maddox red is a group of red cylinders arranged parallel to each other.
- o They produce a red line image from a point source of light (this red line is perpendicular to the axis of the cylinders).
- o The rod is placed in front of one eye and the patient fixates a source of light at distance of (6 metres).
- o An orthophoric (normal) sees the point of light coinciding with the middle of the red line.

3. Maddox wing (for near):

- o The Rt. eye sees 2 arrows (vertical & horizontal).
- o The Lt. eye sees 2 scales (vertical & horizontal).
- o An orthophoric sees the arrows point to zero.
- o In heterophoria: the patient can directly read his angle of squint.

Treatment:

1. Compensated cases:

No treatment (asymptomatic).

2. Decompensated cases:

- Correct any error of refraction.
- Strengthen the weak muscle by:
 - a. Orthoptic exercises: esp. in convergence insufficiency.
 - b. Exercising prisms: increase muscular effort.
 - c. Surgery: if all the above failed (in large degrees, rare).
- Relieving prisms:

Disadvantages: Latent squint becomes manifest.

NB.

Exercising prisms: base towards deviation.

Relieving prisms: apex towards deviation.

4. Paralytic squint and how to diagnose.

Definition:

Manifest squint (eye deviation) due to paralysis of one or more of extraocular muscles.

Etiology:

Due to lesion in the muscle or its nerve supply (lower motor neuron lesion).

1. Nuclear lesions:

- Congenital: absence of nucleus.
- Inflammatory: encephalitis, brain abscess.
- Vascular: thrombosis, hemorrhage or embolism (the).
- Neoplastic: Tumors of brain stem (midbrain & pons).
- Other (toxic): as in diphtheria, alcohol & lead.

2. Nerve lesions:

- Congenital.
- Inflammatory: meningitis, cavernous sinus thrombosis.
- Trauma: fracture base of skull.
- Neoplastic: brain tumor, orbital tumor.
- Vascular: hemorrhage (subdural or subarachnoid) & cavernous sinus thrombosis.

3. Muscle lesions:

- Congenital: maldevelopment of the muscle (hypoplasia).
- Trauma: contusion or hemorrhage in muscle sheath or fracture orbital bones.
- Neuromuscular: myopathy & myasthenia gravis.
- Neoplastic: orbital tumors.
- Thyrotoxic exophthalmos.

NB.

- Ophthalmoplegia:

External: paralysis of EOM.

Total: paralysis of both extra and intra ocular muscles (EOM & IOM).

- Superior orbital fissure syndrome:

Includes affection of 3rd, 4th, 6th & 1st division of 5th (ophthalmic branch of trigeminal).

- Orbital apex syndrome:

Includes SOF syndrome + optic nerve affection.

Clinical picture&diagnosis:

1. *Ocular deviation:*

The paralyzed muscle loses tone and antagonist muscle draws the eye towards it (in opposite direction of action of paralyzed muscle).

2. *Angle of deviation:*

A. *Primary angle of deviation:*

- o Angle of squint when the patient is fixing with the normal eye.
- o Diagnosed by corneal light reflex.

B. *Secondary angle of deviation:*

- o It's the angle of squint seen when the patient is fixing with the squinting eye. i.e. The normal eye is covered.
- o N.B. In paralytic squint, 2ry angle > 1ry angle.

Explanation:

- o Normally, impulses reaching the 2 eyes in order to look at any direction are equal to both eyes (yoke muscles = Herring law).
- o When paralyzed eye is fixing (normal eye is covered), more impulses reach paralyzed muscle to force it to contract.
- o At the same time, normal eye (synergistic ms.) will receive equal amount of impulses.
- o Thus, normal eye will deviate much more under cover (larger angle).

3. *Binocular diplopia:*

- Cause:

- o Image falls on non corresponding retinal points.
- o Image seen by normal eye is clear as it falls on fovea = true image.

o Image seen by squinting eye is blurred as it falls outside the fovea = false image.

- Characters:

1. Maximum when the patient looks at object situated in same direction of action of paralyzed muscle.
2. Disappears when one eye is covered.

- Maybe:

1. Uncrossed (homonymous) diplopia:

False image falls on same side of paralyzed eye (as in LR paralysis).

2. Crossed (heteronymous) diplopia:

False image falls on opposite side of paralyzed eye (as in MR paralysis).

- Patient fights diplopia by:

1. Suppression (if a child) then amblyopia.
2. Compensatory head posture.
3. Covering one eye.

4. *Compensatory head posture:*

Abnormal head positions to minimize diplopia. The head is turned in same direction of action of the paralyzed muscle.

5. *False projection (past-pointing):*

False orientation of objects situated in same direction of action of paralyzed muscle (wrong estimation of the site when the normal eye is covered).

Cause:

Brain sends excessive impulses to the paralyzed muscle to force it to contract.

6. *Headache, nausea & vertigo:*

Causes:

Diplopia & false projection.

Complications:

1. Suppression (in children below 9 years):
Suppression of false image to avoid diplopia leading to amblyopia.
2. Muscle changes (in older age):

- Direct antagonist: overaction due to contracture (on prolonged palsy).
- Indirect synergist: overaction due to excessive impulses.
- Contralateral antagonist: underaction due to inhibitory impulses.

Diagnosis:

1. Testing ocular motility: ask the patient to fix your finger in 6 cardinal directions.
2. Diplopia chart.
3. Hess screen.
4. Cover test: no movement.

Treatment:

1. Treatment of the cause:
 - May lead to complete recovery (spontaneous nerve regeneration).
 - During that: cover the squinting eye to avoid binocular diplopia.
2. Surgical treatment:

Required if no recovery occurs after 6 months of ttt.

- a. If the muscle is weak (paralyzed ms. crosses midline = paresis):

Strengthen it by resection & weaken its direct antagonist (recession).

- b. If the muscle is completely paralyzed (can't move to midline):

Transposition of healthy non paralyzed muscle (Jensen operation, only in lateral rectus paralysis).

<i>3rd nerve palsy</i>	<i>4th nerve palsy</i>	<i>6th nerve palsy</i>
Ptosis (levator paralysis).	Eye deviates up.	Convergent squint.
Paralysis of EOM + deviation out (lateral rectus).	Diplopia increases on looking down.	Diplopia (uncrossed).
Paralysis of accommodation (ciliary muscles).		
No diplopia (due to ptosis).		
Pupil dilation (paralysis of constrictor pupillae).		
Mild proptosis (loss of tone of paralyzed muscle).		

5. Describe clinical picture and investigations of left lateral rectus palsy.

It's due to paralysis of left lateral rectus muscle due to injury of left abducent (6th) nerve.

Symptoms:

1. Left convergent squint = esotropia (angle of squint is larger on looking to the left).
2. Limited ocular motility to the left.
3. Binocular diplopia (uncrossed & increased on looking to the left).
4. False projection on looking to the left.
5. Face turn to the left.

Diagnosis:

As before:

1. Corneal light reflex.
2. Angle of deviation:
Primary and secondary.
3. Compensatory head posture.
4. Other tests:
 - a. Diplopia chart.
 - b. Hess chart.
5. False projection.
6. Ocular motility:
Limitation of abduction of left eye.

Investigations:

1. Send and refer to neurologist.
 2. MRI brain to exclude tumors or aneurysms.
-

6. Concomitant squint.

Definition:

A manifest squint which is not due to paralysis of extraocular muscles and in which angle of squint is the same in all directions of gaze (primary and secondary).

Etiology:

- Binocular vision is fully developed between the ages of 6 months and 6 years.
- Any obstacle to binocular vision during this critical period results in ocular deviation.

Obstacles maybe:1. Refractive (uncorrected error of refraction):

a. In hypermetropia (more than 3D):

- Child accommodates to see clearly.
- Accommodation is associated with convergence = esotropia.

b. In myopia:

Relaxation of accommodation & lack of convergence = exotropia (concomitant convergent squint).

2. Non-refractive:

a. Congenital:

Esotropia is more common than exotropia:

- Peripheral motor obstacle: any anomaly of EOM.
- Central obstacle: defective development of presumed fusion center or visual pathway.

b. Sensory obstacle:

- Due to monocular impaired vision (as unilateral cataract).
- If visual acuity in one eye is weak, the brain will suppress it (unilateral squint).

Sequelae of concomitant squint:

- With onset of squint, images of the object will fall on non corresponding retinal points leading to binocular diplopia.
- But, as the patient is still young, the brain finds a solution for this and diplopia doesn't persist.

Solutions are:1. Suppression (more common):

- Active neglect by the brain for image seen by squinting eye.
- Temporary and occurs only when non-squinting eye is fixing.
- If suppression is permanent, it's called amblyopia.

2. Eccentric fixation:

- Occurs in some cases of amblyopia.
- Patient develops the ability to fix objects by a part of the retina other than the macula (false macula).
- It's due to dense suppression of macula.
- On covering the fixing eye, squinting eye fix the object without any movement and with bad visual acuity (6/60).

Clinical picture:

Symptoms:

Deviation of one or both eyes.

Signs:

1. Test ocular motility in 6 cardinal directions to exclude paralytic squint.
2. Measure visual acuity:
 - a. If equal in both eyes = alternating squint (patient can fix by both eyes).
 - b. If poor in squinting eye = unilateral squint.
3. Cover-uncover test:
 - a. Ask the patient to fix an object (1st at a distance, then near).
 - b. Cover the fixing eye:
 - c. The squinting eye will fix and the fixing eye will deviate.
 - d. Remove the cover:
 - If the new position remains: it's alternative squint (suppression).
 - If the original position returns: it's unilateral squint (amblyopia).

NB. No movement occurs in:

- Eccentric fixation (bad V.A.).
- Apparent squint (good vision).
- Paralytic squint.

- Blind eye (no P.L.).

4. Measurement of angle of squint:

A. Corneal light reflex (Hirschberg test):

- Depends on 1st purkinje image.
- Hold the torch in front of patient's nose.
- Observe the light reflex on the cornea of squinting eye.
- Rough test.

B. Synaptophore (major amblyoscope):

- Composed of 2 tubes which can be moved on a graded scale.
- Ask the patient to look through the tubes. Then ask him to rotate these tubes till the 2 test objects (at tube end) are superimposed (bird inside the cage).
- Angle between the two tubes = angle of squint.

C. Prism cover test (Krimsky test):

Angle of squint nearly equals prism power that abolishes movement on cover test.

5. Assessment of binocular vision:

Definition:

Co-ordinated use of the two eyes to produce single mental impression.

Values:

- Stereopsis (depth perception).
- Larger field.
- Masks possible field defects in the other eye.

Grades:

a. Simultaneous macular perception:

Ability to perceive and superimpose the images of the 2 maculae.

b. Fusion:

Ability to fuse 2 images into 1 complete image.

c. Stereopsis:

Depth perception.

Assessment:

- a. Synaptophore.
- b. Worth's four dots test:
 - Ask the patient to wear red-green goggles.
 - Ask him to look to the 4 colored illuminated dots.

Results:

- Binocular vision = 4 dots.
- Diplopia = 5 dots.
- Rt. suppression = 3 green dots.
- Lt. suppression = 2 red dots.

*Treatment:*Aim:In a child:

1. Build and restore binocular vision.
2. Improve and preserve visual acuity in squinting eye.
3. Improve cosmetic appearance.

In adult:

Improve cosmetic appearance.

Time:

As early as possible (before age of 7 years) to:

1. Avoid amblyopia.
2. Allow binocular vision to develop.

Lines of ttt:

1. Atropine eye ointment: for accommodative squint in children < 2 years till they can wear glasses.
2. Glasses: correction of error of refraction, as soon as the child can wear them.
3. Amblyopia therapy: covering the normal eye to improve vision in amblyopic eye.

Plan of treatment:

Adult:

Surgical correction of squint (muscle resection or recession) for cosmetic purpose only.

Child:If refraction error is matching with type of squint:

1. Glasses:

- If successful, do orthoptic ttt.
- If not successful:

2. Occlusion therapy:

- If successful, do orthoptic ttt.
- If not successful:

3. Squint surgery, followed by orthoptic ttt.

If error of refraction is not matching with type of squint:

Do squint surgery followed by orthopticttt.

7. Compare between concomitant and paralytic squint.

	<i>Concomitant</i>	<i>Paralytic</i>
<i>Definition</i>	See before.	See before.
<i>Ocular motility</i>	Free.	Limited.
<i>Angle of squint</i>	Constant (primary = secondary).	Secondary > primary.
<i>False projection & compensatory head position</i>	Absent.	Present.
<i>Diplopia</i>	Absent due to suppression.	Present.

Rest of differences: see before.

8. Benefits of cover test.

1. Latent squint: diagnosis.
2. Paralytic squint: proves that secondary angle of deviation is larger than primary angle.
3. Concomitant: differentiation between unilateral & alternating squint. + Diagnosis of eccentric fixation.

9. Define diplopia, mention its types & causes.

Definition:

Simultaneous perception of two images (instead of one) for the same object by both eyes.

Types:

1. Uniocular (monocular), appears on covering normal eye.
2. Binocular.

Causes:

Binocular diplopia: stimulation of non corresponding retinal points.

<i>Uniocular diplopia</i>	<i>Binocular diplopia</i>
Early cortical cataract.	Paralytic squint.
Subluxated lens.	Decompensated latent squint.
Irido-dialysis.	Proptosis (if unequal or unilateral).
Anisometropia (corrected by glasses).	Symblepharon & pterygium.
	Displaced globe (by orbital tumor).
	Anisometropia (if corrected by glasses).

MCQ:

1. All of the following muscles arise from annulus of Zinn, except:

- a. Inferior rectus.
- b. Inferior oblique.
- c. Superior oblique.
- d. Superior rectus.

2. The oculomotor (3rd) nerve supplies all of the following except:

- a. Inferior oblique.
 - b. Inferior rectus.
 - c. Superior oblique.
 - d. Superior rectus.
-

3. 2ry angle of deviation is greater than 1ry angle of deviation in case of:

- a. Latent strabismus.
 - b. Paralytic strabismus.
 - c. Concomitant strabismus.
 - d. None of the above.
-

4. Monocular diplopia can be due to:

- a. Paralytic squint.
 - b. Blow out fracture of orbit.
 - c. Iridocyclitis.
 - d. Iridodialysis.
-

5. For investigation of latent strabismus, the following test is diagnostic:

- a. Cover-uncover test.
 - b. Hess screen.
 - c. Detection of limitation of ocular motility.
-

Intra ocular Tumour

GIVE AN ACCOUNT

1. Discuss retinoblastoma:

Definition:

Intra ocular tumor originated from retinoblasts (primitive photoreceptor cells).

Incidence: most common primary IOT in children

- **Age:** less than 3 years.
- **Sex:** Equal.
- **Laterality:** bilateral/ multifocal (30%).
- **FH:** 6% due to mutation in chromosome 13 (loss of antioncogene).

Symptoms:

- 1) Quiescent stage:
 - Leucocoria (60%): Amarotic cat 's eyes.
 - Squint (20%): if central at fovea.
 - Asymptomatic: if symptomatic and peripheral.
 - 2ry uveitis (masquerade syndrome).
- 2) Secondary buphthalmous.
- 3) Extraocular intraorbital stage: proptosis.
- 4) Distant spread → optic nerve → CSF → Brain.

Signs: (Fundus)

- Endo-phytic: white with surface vascularization and calcium (**Cottage cheese apperence**).
- Exo-phytic: 2^{ry} exudative RD.

Investigation:

- 1) US (most important), CT scan, MRI, FA, Doppler.
- 2) Of the spread:
 - Lumbar puncture 'CSF'
 - MRI brain

N.B: Avoid CT/X-ray in genetic type for the risk of 2^{ry} malignancy e.g. osteosarcoma.

Treatment:

1) Quiescent stage: Tumour destruction:

- If small:
 - Cryo. (anter.)/ laser photo-coagulation (Post.)/ **Transpupillary thermotherapy** which is applying heat to the tumour by using a diode laser that leads to cellular destruction and inhibition of DNA synthesis so dec. in size.
- If Large:
 - **Radio-therapy:** brachy (sclera explants) or teletherapy.

2) Glaucomatous stage: Enucleation not eversion.

3) Extra-ocular stage: Exentration.

4) Distant spread: Palliative.

N.B:

1. **Examine both eyes and family member < 6years**
2. +/- chemo-reduction.
3. **If enucleation is to be done:**
 - Take the longest stump of the optic nerve.
 - Examine the edge of the tumour.

2. D.D of leucocoria: (v.v.v.imp.):

- 1) Retinoblastoma (M.S.)
- 2) Congenital cataract. (M.C.)
- 3) Retinopathy of pre-maturity.
- 4) Cylitic membrane.
- 5) Coats disease
- 6) PHPV

3. Discuss Malignant Melanoma:

Definition:

Intra ocular tumor originated from the uveal melanocytes (commonest: choroid > CB > Iris).

Incidence: most common primary IOT in adults

- **Age:** more than 50 years.

- **Sex:** more in males.
- **Laterality:** unilateral/unifocal.
- **FH:** more in blacks.

Symptoms:

- 1) Quiescent stage:
 - Field defect if peripheral and large.
 - Drop of VA: if central.
 - Asymptomatic if small and peripheral.
 - 2^{ry} Uveitis.
- 2) Glaucomatous (2^{ry}) stage.
- 3) Extraocular intraorbital: Proptosis in choroidal melanoma and pigmented epibulbar mass in CB melanoma.
- 4) Distant spread: blood metastasis: liver-lung-long bones.

Signs:

Subretinal: Dome pigmented mass (rarely amelanotic) with surface lipofuscin orange pigments that when ruptures the Bruch's membrane → Mushroom (collar stud) shaped → 2^{ry} exudative RD.

N.B: Gonioscopy in iris and CB melanoma.

Investigations:

- 1) US (most imp.),CT,MRI,FA,Doppler.
- 2) Of spread:
 - Liver enzymes
 - CT chest and abdomen.
 - Bone Scan.

Treatment: as retinoblastoma.

D.D:

- 1) Choroidal navus/hemangioma.
- 2) Iris navus (flat and small).
- 3) Metastasis (breast-bronchus),(usually bilateral +/- multifocal).
- 4) Other causes of RD e.g: Rhegmatogenous/TRD.

MCQS:

1. Malignant melanoma of the choroid is characterized:

- a) a. Producing true retinal detachment
- b) b. producing secondary glaucoma.
- c) c. Appearing mushroom-shaped in US.
- d) d. Occurring in infants with family history of similar conditions.

Answer: (C) Appearing mushroom-shaped in US.

2. Amaurotic cat's eye reflex, in all except :

- a. Retinoblastoma.
- b. PHPV.
- c. Coat's disease (retinal telangiectasia)
- d. Toxocara.
- e. MM Choroid.

ANSWER: (E)

Laser

ESSAY QUESTIONS:

1. Uses of laser in ophthalmology.

1. *Lid disorders:*

Removal of warts and papillomas (CO₂ laser is used).

2. *Errors of refraction:*

Excimer laser photo-ablation (LASIK & PRK) is widely used to correct a wide range of refractive errors.

3. *Cataract:*

- YAG laser capsulotomy to treat late postoperative posterior capsule opacification following cataract surgery.
- Erbium-YAG laser is used to remove the cataractous lens.

4. *Retinal disorders:*

Argon or diode laser maybe used to seal retinal tears, treatment of diabetic retinopathy & retinal vascular disorders.

5. *Glaucoma:*

- Laser iridotomy using YAG laser.
- Argon or diode laser trabeculoplasty in some cases of open angle glaucoma.
- Scanning laser ophthalmoscopy to visualize optic nerve head in cases of glaucoma.

2. Complications of laser in ophthalmology.

1. Opacification of cornea, lens or vitreous.
 2. Hemorrhage: hyphema, vitreous hemorrhage or retinal hemorrhage.
 3. Foveal damage: in uncooperative patients.
 4. Increased IOP (transient).
-

CASES:

1. A 25-year-old myopic male came to the eye clinic complaining of rapidly progressive loss of the lower field vision in his right eye. The condition had started 2 days earlier. one week earlier he had received trauma to his head. A couple of days after the head trauma he noticed the appearance of dense floaters in his right eyes as well as flashes of light. The attending ophthalmologist should proceed to: (Retina)

- 1- Measure the degree of his myopia and prescribe a new pair of glasses.(False)
- 2- Start to give him anti-glaucoma treatment to manage his field defect.(False)
- 3- Immediately examine the fundus of both eyes with maximum mydriasis.(True)
- 4- Do him a panretinel photocoagulation using Argon laser.(False)
- 5- Excimer laser is the best line of treatment in this case.(False)

2. A patient with the history of a retained intra-ocular foreign body in his right eye, since 5 years, that has not been removed due to poor prognosis presented with pain and tenderness in both eyes and diminution of vision in his left eye.(Injuries)

- 1- The condition should be managed urgently with systemic and topical steroids.(True)
- 2- Diffusion of iron particles from the foreign body is the cause of the present condition.(False)
- 3- The condition is caused by immune mediated reaction .(True)
- 4- The condition would have been avoided by enucleation of the right globe immediately after trauma (True)
- 5- The present condition in the left eye is unrelated to the right eye(False)

3. A mother brought her 2 years old boy to the eye clinic saying that she has recently started to notice that his eye become deviated inwards. She has also noticed a white coloration in the pupillary area of his right eye. The boy was born was born at full term and was not placed in an incubator immediately after birth. The boy's father had one of his eyes enucleated since he was an infant. The following steps should be done

by the attending ophthalmologist:(Tumours)

- 1- Re-assure the mother and tell her that there is nothing serious and send boy home.(False)
- 2- Do refraction for the boy with atropine eye drops and prescribe him eye glasses to correct the squint.(False)
- 3- Request an immediate fundus examination of both eyes under general anesthesia with full mydriasis(True)
- 4- Request CT scans for head and orbit(True)
- 5- Warn the mother that there is a possibility for the same condition to affect future children.(True)

4. A sixty-five years old lady visited her eye doctor to have her glasses checked and to check her fundus. By the time she came home she started to have coloured haloes. Followed shortly by severe right temporal headache and vomiting. (Glaucoma) answer the following questions by true or false:

- 1- Anterior chamber of the right eye is expected to be deep (False)
- 2- The cornea is expected to be hazy.(True)
- 3- Treatment of this case is essentially medical.(False)
- 4- Atropine drops should be started immediately(False)
- 5- Topical steroids can be used in the management of this case(True)

5. A twenty-year-old tennis player received a severe trauma to his left eye by a tennis ball. The second day, after subsidence of oedema, he noticed that he has double vision.

Slit lamp examination of the left eye showed that the anterior chamber was irregular, pupil semi dilated and fixed and the edge of the crystalline lens was seen to transverse the pupil.(Subluxation)

Answer the following questions by true or false

- 1- Double vision will disappear by covering the right eye (False)
- 2- Irregular astigmatism can result from this condition.(True)

- 3- In some such cases, visual acuity can be improved by a convex lens.(True)
- 4- The retina in this case is expected to show micro aneurysms.(False)
- 5- Surgical management is not an option here.(False)

6. A 38 years old pregnant lady developed severe hypertension and lower limb oedema. She noticed painless blurring of vision of both eyes.

Answer the following questions by true or false:(Retina)

- 1- The optic nerve head is expected to show deep cup (False)
- 2- Retinal exudates in this case are typically cotton wool.(True)
- 3- If retinal detachment develops in this case, the first area to be affected is the upper temporal retina.(True)
- 4- If retinal detachment develops, immediate retinal surgery is indicated.(False)
- 5- In the absence of retinal detachment, conservative follow up is indicated till the expected date of delivery.(True)

7. A sixteen-year-old boy complained of right eye inflammations that started since few days. Slit lamp examination of this eye showed a dull white limbal nodule at five o'clock measuring about one half mm. surrounded by blood vessels (Conj.)

Answer the following questions by true or false:

- 1- The treatment of this nodule is basically by antibiotics and atropine. (False)
- 2- This lesion may be related to TB (True)
- 3- Corneal perforation may result from this condition (False)
- 4- This lesion may develop into a pannus that is crescent shaped with a serrated border(False)
- 5- Treatment of this lesion gives a permanent immunity against recurrence(False)

8. A fifty-five-year-old male complained of flashes of light after a minor trauma to his left eye. A week he noticed that the vision of this eye is

severely diminished even with his -8.0 diopter glasses. The intraocular pressure of his eye was 9.0 mmHg. The light reflex coming from his pupil was grey.(Retina)

Answer the following questions by true or false

- 1- The pupil of his eye is expected to be dilated with loss of direct and consensual reaction to light(False)
- 2- Fluorescein angiography is the investigation of choice that can be used for the left eye (False)
- 3- Excimer laser is the type of laser suitable for treating the left eye (False)
- 4- A vitrectomy procedure may be performed to treat this eye(True)
- 5- Family history plays a role in this disease(True)

9. A mother came in complaining that her newly born boy has a constant watering of both eyes. Both eyes looked big on provisional examination. The large diameter cornea of the right eye was cloudy, while that of the left eye, though large in diameter, was clear. The anterior chamber of the left eye could be seen to be very deep. The white of both eyes looked bluish.(Glaucoma)

Answer the following questions by true or false

- 1- This condition resulted from maternal birth tract infection (False)
- 2- The iris can be tremulous(True)
- 3- Treatment should be postponed till the age of child is 6 months(False)
- 4- The treatment is essentially medical for the left eye(False)
- 5- Surgery is treatment of choice for both eyes(True)

10.A young man who received a head injury reported that, although vision in both eyes was unaffected, yet he could see an additional blurred image to the side of the clear image and in order to avoid this annoying image he had to turn his face always to the left side (Injuries)

- 1- Immediate surgery is needed to correct his complaint (False)

- 2- The annoying blurred image is expected to disappear if he covers his right eye(True)
- 3- Prisms may play a role in the management of this case(true)
- 4- Would removal of the crystalline lens treat this condition (False)
- 5- Ultrasonography is the proper investigation needed in this case(False)

11.A 45-year old male patient presented to the clinic with ocular pain, headache, lacrimation, photophobia and diminution of vision. On examination the lids were edematous, the conjunctiva injected but no discharge was found. Anterior chamber showed flare and cells, the iris was indistinct and the pupil miotic. IOP was 14 mmHg and fluorescein test was negative.(Uvea)

- 1- Miotics are the 1st line of treatment(False)
- 2- Steroids are beneficial(True)
- 3- Clogging of the angle in the early stages causes secondary glaucoma (True)
- 4- Rheumatoid arthritis is a common association of this condition (True)
- 5- Chest X-Ray is not one of the investigations needed to diagnose the etiology of this condition (False)

12.A 55-year old diabetic and hypertensive female came complaining from abrupt drop of vision in the right eye. She gives history of having received laser sessions in her left eye a while ago.(Retina)

- 1- Complicated cataract is the 1^{ry} cause of this patient complaint.(False)
- 2- This patient may be suffering from renal problems. (True)
- 3- Poor glycemic control is not related to this condition. (False)
- 4- Ultrasonography is a good tool to evaluate the condition of the right eye. (True)
- 5- Neovessels in the retina could be the cause of the drop of vision in the right eye.(True)

13.A 19-year-old man was complaining of frequent change of glasses, his refraction was -6.0 D spheres with -4.0 cylinder in the right eye. Central corneal linear opacities were evident on slit lamp examination of the

left eye. Pachymetry showed decreased corneal thickness in both eyes (Errors)

Answer the following questions by true or false

- 1- Soft contact lenses can correct the error on his right eye.(False)
- 2- The vertical linear opacities are termed Haab'sstriae(False)
- 3- LASIK is a safe alternative to glasses.(False)
- 4- Vernal conjunctivitis is a common association.(True)
- 5- Penetrating keratoplasty (PKP) has a low success rate in this patient's left eye.(False)

14.A 28-year-old man presented with right painful red eye accompanied with diminution of vision. patient mentions recurrence of this condition twice before in the past 5 years. Physical examination revealed the presence of aphthous ulcer on the mouth, tongue and genitalia.(Uvea)

Ophthalmological examination:

- **Vision in the right eye: CF at 3 meters**
- **Anterior segment: ciliary injection**
- **Keratic precipitates on the back of the cornea**
- **Anterior chamber: aqueous cells and flare**
- **IOP was within normal**

Answer the following with true or false:

- 1- The pupil is usually miotic in this condition (True)
- 2- Pilocarpine eye drops are used for the management(False)
- 3- The disease is usually infectious in origin(False)
- 4- The anterior chamber may show hyphema(True)
- 5- The retina may be affected in this condition(True)

15. A 24-year-old obese woman presents with an increasing frequency of headache during the past 2 months. Headaches were worse in morning and are associated with nausea and sometimes projectile vomiting. She also notes transient attacks of blurring of vision. She denies systemic illness. She has been taking contraceptive pills for the past 2 years. (Optic Nerve)

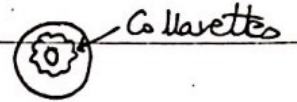
Answer the following with true or false:

- 1- The fundus of this patient may show papilledema (True)
 - 2- Visual acuity is usually markedly affected early in the disease (False)
 - 3- The pupillary reaction to light is expected to be normal (True)
 - 4- Brain CT will help in the diagnosis (True)
 - 5- Antibiotics are the main line of treatment in this case (False)
-

Uvea

① Iris: Ant part of uveal tract → divides Ant. segment → AC
 At birth → blue-gray → definitive Color at one year → PC

* 2 Borders → Central free
 → Attached ciliary



* 2 Surfaces → Ant. → elevations → Collarettes
 → Depressions → Radial streaks
 → Post. → Smooth + dark → Crypts
 → Circular furrows

* 2 parts → Stroma → Pigmented cells
 → Radial blood vessels
 → Nerves
 → Muscles → sphincter
 → dilator
 → post. epith → 2 pigmented layers

* Nerve Supply → Sensory → long ciliary nerves
 → Motor → short ciliary nerves → Parasymp. → 3rd nerve
 → Symp. → Cervical chain

* Blood Supply → Circulus iridis anterioris major → 7 Ant. ciliary art.
 → Circulus iridis vasculosis minor → 2 long Post. ciliary art.

② Ciliary body → triangular base ant. and apex post.

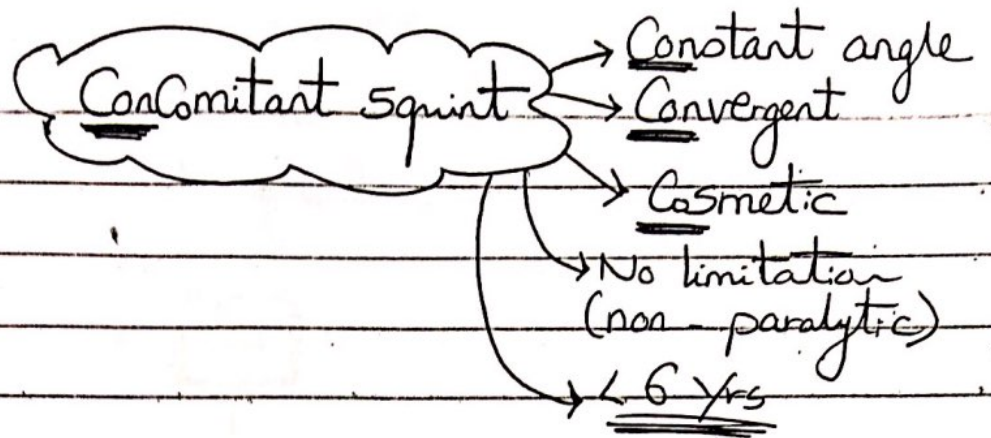
* 2 parts → Pars plicata ($\frac{1}{3}$) ant. → 70 Ciliary Processes → outer non-pig.
 → inner pigment.
 → Pars plana ($\frac{2}{3}$) Post.

* Stroma → Blood vessels
 → Nerves
 → Melanocytes
 → Ciliary muscle → Circular → accommodation
 → Longitudinal → Aq. drainage
 → oblique → bet. both

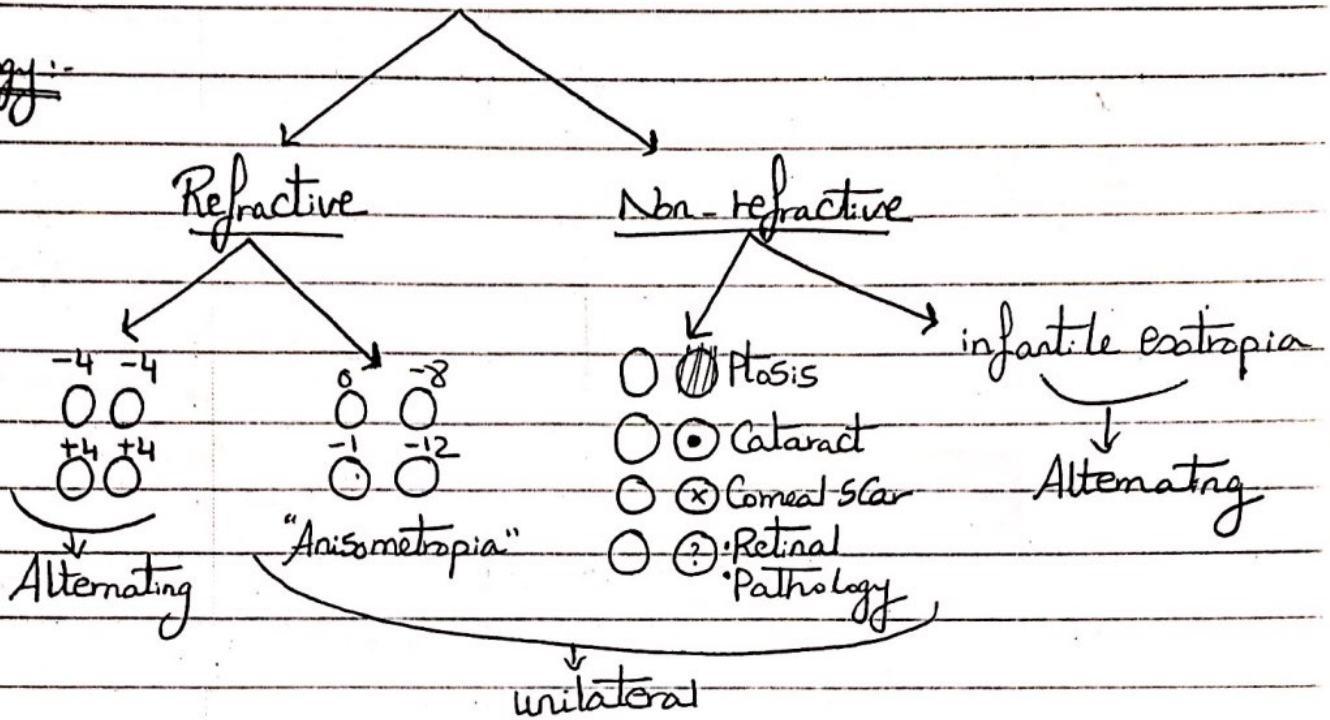
(N.B) Nerve Supply of Choroid is only vasomotor ∴ insensitive to pain

	Endophthalmitis	Panophthalmitis
<u>Def</u>	Acute suppurative inf of middle, inner coats + cavities	As endo + outer coat and Tenon's capsule
<u>Etiology</u>	1) <u>Infective</u> "mainly" :- * Exog. (BP) → Post-operative → Penetrating trauma → perforating ulcer * Endog. (metastatic) :- in immunocompromised patients 2) Non infective :- post operative "sterile" glove powder	As endophthalmitis
<u>CA</u>	1) Acute :- Staph. epiderm. (albus) 2) Delayed → Fungal → propiono bact. acne	As endoph.
<u>Symp</u>	Severe PDR + upto no PL	As endophthalmitis + Pyrexia + No PL
<u>Signs</u>	As uveitis (more severe) Infected hypopyon vit. abscess (yellow reflex)	As endophthalmitis + - proptosis - ophthalmoplegia - perforation
<u>Complic</u>	1) Spread → Panophth. → orbital cellulitis → Brain 2) Healing → Pthisis bulbi	As endoph.
<u>Mt</u>	If no PL → enucleation better than evisceration	Enucleation only NEVER evisceration

	Myopia	Hypermetropia	Regular astigmatism	Irregular astigmatism
1) <u>Optical</u>	- Spherical can cause - Glasses or CL	- Spherical Convex - CL are less tolerated	- Simple → Cylindrical - Compound → Sphero-cylindrical - <u>Mixed</u>	- Rigid gas permeable or hard CL - (Contact Corneal Pave)
2) <u>Surge</u>	* Corneal → PRK → LASIK * Lens → Phakic IOL → Clear lens extraction	- Refractive Corneal LASIK (At the periphery)	* Laser → LASIK * Incisional → Astigmatic Keratotomy * IOL → Toric IOL → Toric ICL	- Keratoplasty of the Cause
3) <u>Prophylactic</u>	- Regular follow up for lattice + holes - Prophylactic laser			
4) <u>Complication</u>	1) Buckle if RD 2) Cataract extraction 3) Glaucoma			



* Etiology:-



* Signs:-

- 1) VA → unequal = unilateral
- 2) External + Slit lamp → Cause
- 3) Retinoscopy → uncorrected error
- 4) Fundus → exclude retinoblastoma (imp)

* Symptoms

- 1) Cosmetic
- 2) No diplopia

5) Corneal light reflex = PAD

6) Cover uncover → SAD = PAD

7) Angle = PAD → pupillary edge = 15
→ limbus = 45

Type → Alternating → العينين بملوا
→ unilateral → العينية زي ماسه

8) Motility → No limitation

9) BNV → Synaptophore or Worth's 4 dot test

ttt of Concomitant Squint

Age

Young age
 < 6 yrs

> 6 yrs

Cosmetic
 ↓
 Surgical

of The Cause

Refractive

Myope
 Full Correction
 Hypermet.
 Total Error
 Anisometria
 - CL

Organic lesion

- Ptosis, IOL...
 "Surgical"

of amblyopia

- Before 6 yrs
 - After ttt of Cause
 - Competitive Occlusion therapy

If residual angle still present
 - orthoptic exercises
 - Surgical
 (Recession or Resection)

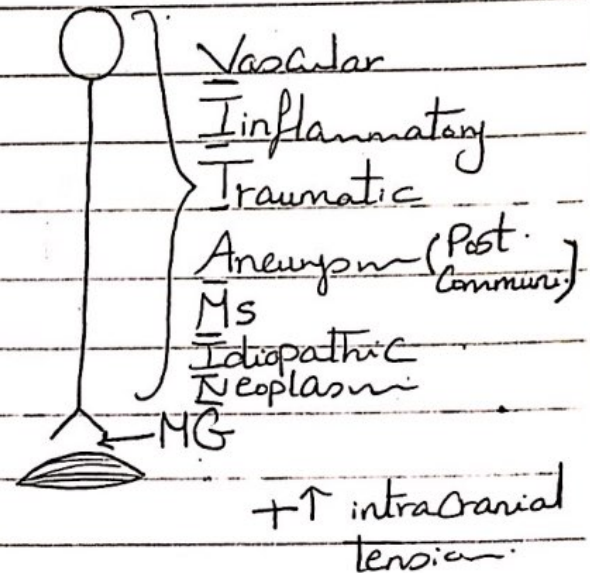
* Sequelae of Concomitant Squint:

- 1) Suppression
- 2) Amblyopia (if permanent suppression)
- 3) Eccentric fixation

paralytic Squint Paresis one EOM
Paralysis more

* Etiology - lower motor neuron lesion of 3rd
4th
6th } and/or

* Types - 3rd → exotropia
6th → esotropia
4th → Hypertropia



* Symptoms - Binocular diplopia

Image falls
on non-
Corresponding
Points

Types

uncrossed

- LR₆
- exotropia

Crossed

- MR₃
- exotropia

vertical

- SO₄
- Hypertropia

* Signs:

- 1) of the Cause: Neurologist - MRI brain
- 2) To avoid diplopia: relieving pressure (apex towards)
- 3) Botox
- 4) After 6 months
 - Paresis → resection
 - Paralysis → Jones operation (transplantation)

VA

External exam

Slit lamp

Retinoscopy

Fundus

reflexes
apex
Neuro
Cus, Cus

Manifest Squint

1) Corneal light reflex → opposite direction of affected muscle

- Esotropia → 6th

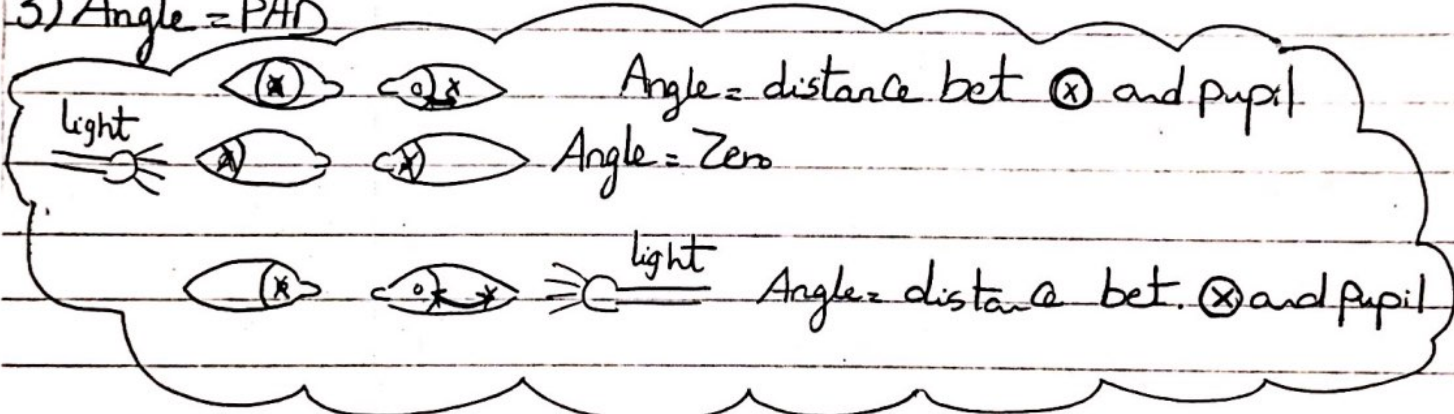
- Exotropia → 3rd

- Hypertropia → 4th

* PAD → variable towards direction of affected muscle

2) Cover-uncover → SAD > PAD

3) Angle = PAD



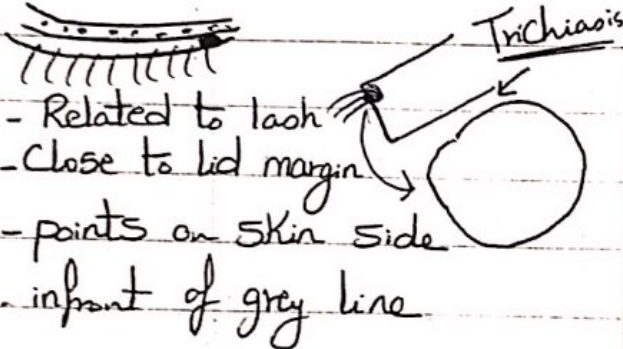
Corneal light reflex (Variable PAD)

- 4) Motility
- 4th → limited depression in adduction
 - 6th → limited abduction
 - 3rd → limited elevation, depression and adduction
 - ptosis → ∴ No diplopia
 - Pupil → dilated inactive

5) Compensatory posture → Horizontal dev. ex: 6th → ipsilat. face turn

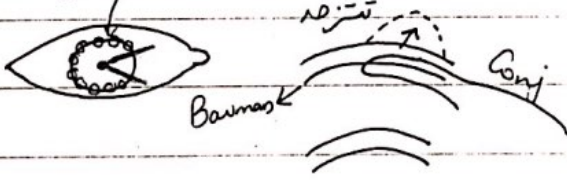
→ vertical dev. ex: 4th → contralat. face turn

lid glands

	Stye (Hordeolum externum)	Chalazion (if inf. → Hordeolum internum)
	Acute → Suppurative Staph. Sub. Sweat glands	Chronic lipogranulomatous Tarsal gland
PDF:	DDM	1) Seborrhea
<u>Case</u>	2) Staph 3) Errors of refraction → shutting eyes → Congestion → Stasis → inf.	2) vit. A deficiency
C/P	 - Related to lash - Close to lid margin - points on skin side - in front of grey line	- Painless - Painful if infected
Complic:	1) Spread → Cavemous Sinus Thrombosis 2) Trichiasis 3) Recurrent → DM, Staph, Errors	1) Mechanical → - UL → Astigmatism - UL → Ptosis - LL → Ectropion 2) Inf. → Acute Chalazion (Hordeolum interna = small abscess) 3) Recurrence * if in old age and same site → Tumor biopsy 4) Marginal Chalazion 5) Multiple Chalazion 6) opens in Conjunctiva 7) Cyst formation 8) Spontaneous resolution

	MPC	PC	Membranes
1) CO :-	Haemophilus egypticus [Koch's weeks bacillus]	Neisseria Gonorrhea	Diphtheria
2) Symp:-	DDR	Severe DDR	DDR
3) Signs:-	Schene	Schene	Schene (woody like) + True membrane
4) Complic:-	Secondary marginal Corneal ulcer.	Secondary Corneal ulcer - Systemic Spread	- Secondary Corneal ulcer - Scar - Systemic Spread
5) ttt:-	BBB not a B	- Hospitalization - Conjunctival Swabs - Systemic Cephalosporin + BBB not a B	- Bed rest - Notification - Anti-diphtheric antitoxic Serum - Glass rod ointment to avoid Sympblepharon.

Conjunctival degenerations

	Pterygium	Pingecula
Def.	Fibrovascular, triangular	Elastotic hyaline degenerations of subepithelial Conj. by UVR in old age
Etiology:	UVR - limbal stem cells deficiency - Elastotic degeneration of stromal collagen + a layer of thick Conj + destruction of Bowman's memb.	- UVR
Symptoms	DD - Disfigurement - Drop of vision $\rightarrow$ $\begin{cases} \text{Papillary effect} \\ \text{Astigmatism} \end{cases}$	
Signs	- Nasal, Bilateral - Apex towards Cornea * 3 parts $\rightarrow$ $\begin{cases} \text{Apex (Stocker's line)} \\ \text{Neck (at limbus)} \\ \text{loosely adherent to sclera} \end{cases}$ 	- Nasal, bilateral - yellow, raised, non-vascular - Base towards Cornea
Types :-	- progressive $\rightarrow$ vascular - Stationary $\rightarrow$ less vascular	
ttt:-	1) Bare sclera $\rightarrow$ $\uparrow$ recurrence 2) Beta irradiation $\rightarrow$ Scleral necrosis 3) Mitomycin C 4) Conj graft 5) Limbal stem cell transplantation	<u>Non</u> unless large or disfiguring

Orbital Cellulitis			
1) Def	- Acute diffuse suppurative of orbital soft-tissue.	- Thrombo-phlebitis of cavernous sinus	- Dys thyroid ophthalmopathy
2) Incidence	mc cause of unilateral proptosis in children.		- Eye and orbital changes of thyroid dysfunction cause of unilateral and bilateral proptosis in adults.
3) Etiology:-	usually infective:- • CA → Bacterial (mc) → Fungal • Source:- → Sinusitis → ethmoidal → Trauma → Buckle, squinting → Spread → Penetrating → Rupture → broad base	- It infection in dangerous area of face - lid → sty - orbit → cellulitis - lacrimal → acute dacryocystitis - Globe → Panophthalmitis	• Autoimmune disease (IGG) • EOM enlargement (infiltrative stage) • Fibrosis (Restrictive stage) - inf. rectus → limited upgaze - MR → limited abduction - SR → defective depression - LR → defective adduction
4) Clinical Picture:-	- General → Fever + ANH - ocular → Swelling + Severe Pain + Hot and tender	- General (Brain) → Severe illness, drowsiness, delirium, disturbed consciousness - ocular → Swelling + Severe Congestion + other eye affection (6th N palsy, diplopia) + Haemoid edema	- General (Thyroid) → Tremors, Polyphagia, loss of weight - ocular (See p.162) + Swelling
5) Complications	1) Spread <u>Optic neuritis</u> → CST → Panophthalmitis → CRD 2) Exposure Keratitis 3) Localization → abscess + fistula 4) Healing (Fibrosis) → frozen orbit Enophthalmos + restricted motility	1) Exposure Keratitis 2) 25% Mortality rate (Brain abscess - Septicemia - Meningitis)	1) Exposure Keratitis 2) Compressive optic atrophy 3) 2nd Glaucoma
6) Inv.	1) CBC → leucocytosis 2) CT → Sinusitis, FB, Abscess localization	MRV with Contrast	1) Thyroid Function → high, normal or low 2) CT or MRI → EOM enlargement 3) Thyroid 2) Corneal exposure 3) Infiltrative → Steroids + immunosuppressive 4) Surgical → orbital decompression or resection
7) Mx.	<u>Haha</u> - Hospitalization - Anti-biotics - Hot fomentations - Analgesics	<u>Haha</u> - Hospitalization - Anti-biotics - Heparin	

Proptosis

1) Congenital

→ Dermoid Cyst

→ Meningo-encephalocele

→ Neurofibromatosis type 1

→ Pulsating

2) Acquired

→ Endocrine

→ Dysthyroid

→ Traumatic

→ Retrobulbar hematoma

→ Anaesthesia

→ Surgical emphysema

→ SC air

→ Crepitus

→ Carotid Car. fistula

→ Fracture base

→ Pulsating

→ bruit

→ Thrill

→ Plegia

→ Excess disc edema + dilated veins

→ Proptosis ↓ by ipsilateral Carotid Compression

→ Inflammatory

→ Acute inf

→ Cellulitis

→ Pan

→ CST

→ Chronic TB

→ Non-infective idiopathic

→ Dacryoadenitis down + in dystopia

→ vascular

→ Orbital varices

→ intermittent

→ ↑ by ↑ venous Pressure

→ Blood Cyst

→ Aneurysm

→ CCF - CST

→ parasitic

→ Hydatid cyst

→ Bone disease

→ Paget's

→ Neoplastic

→

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